A

# LABORATORY TEXT-BOOK

OF

# PATHOLOGY

FOR THE USE OF

## STUDENTS AND PRACTITIONERS OF MEDICINE

BY

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With One Hundred and Twenty-one Illustrations



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TO

LEWIS A. STIMSON, M.D.,

PROFESSOR OF SURGERY IN THE UNIVERSITY MEDICAL COLLEGE OF NEW YORK;
SURGEON TO THE NEW YORK HOSPITAL AND TO THE
HUDSON STREET HOUSE OF RELIEF,

This Volume is Respectfully Dedicated,

IN TOKEN OF HIGH APPRECIATION OF HIS GREAT KINDNESS AND
OF HIS INVALUABLE INSTRUCTION DURING A SERVICE AS
HOUSE SURGEON TO THE NEW YORK HOSPITAL,

BY THE AUTHOR.

# PREFACE.

The aim of this volume must be stated in clear terms in order that its limitations may be definitely understood, and that it may not go into the hands of the critic as a complete treatise on pathology. For the medical student the complete text-books of pathology are too full in their text, and more time is required for its mastery than the student can give to this one branch. Furthermore, in the most thorough laboratory courses given by the colleges, only the important pathological lesions are demonstrated, and the student must search through the complete treatise on pathology for the lesion he is studying. The aim of the present volume is to furnish the student with a text-book that he can have beside his microscope in the laboratory; a book that gives him a concise and accurate idea of the lesions, is brief in its text, yet omits none of the important pathological lesions, nor the mention of any part in a given tissue change.

The method of illustrating by photomicrographs was adopted in order that the student might see in the diagram of the specimen to be studied a picture, not of clear-cut lines, diagrammatic arrangement, and magnified clearness of the point to be illustrated, but the *actual* picture that is given by the specimen under his microscope. In some cases it has been impossible to take a good photomicrograph of the tissue described, and drawings have been introduced. It is a point of great regret to me that the number of these diagrams must be limited by the exigencies of publication.

The author wishes to express his hearty thanks to Dr. M. A. Brown for his careful reading of the manuscript, and for many

valuable suggestions which have added much to the value of the work; to Dr. L. J. Krouse for the specimens used in figures 12, 13, 16, 36, and 97; to Dr. D. I. Wolfstein for specimens used in figures 7 and 8; to P. Blakiston, Son & Co. for a uniform courtesy and care in the work of publication.

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2120 AUBURN AVE., CINCINNATI, OHIO.
*November 12, 1897.*

# CONTENTS.

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OF

# PATHOLOGY.

## INFLAMMATION.

Inflammation is the name given to that pathological condition characterized by redness, swelling, increased local heat, pain, and altered functions in the tissues so affected. It is essentially a series of local tissue changes, combined with pathological exudations from the blood-vessels, and in chronic inflammations with the production of new cells and tissues. It is the response of a living tissue to an injury, be that injury mechanical, chemical, electrical, or parasitic in its nature, provided that its action be sufficient to cause circulatory disturbances with exudation, yet insufficient to cause a death of tissue or an arrest of circulation.

I. **Exudative Inflammation.**—Before the stage of actual inflammation sets in there is always an active hyperemia of the tissues, the blood-vessels are dilated, and the blood-current is in consequence more rapid on account of the lessened resistance. There quickly supervenes a pathological modification in the structure of the walls of the blood-vessels themselves, and a slowing of the blood-current even to stagnation, accompanying or following which an exudation from the blood-vessels takes place, owing to an increased permeability of their walls. There also occurs an increase in the number of the polynuclear leukocytes in the blood of the inflamed organ, and these leukocytes rapidly collect in the peripheral plasma zone of the smaller veins. Here the cells roll along the vessel wall, become adherent, and are dragged into various shapes by the blood-current, or collect in marked accumulations. The important element in the pathological alteration of the vessel wall is a softening and widening of the cement lines between the

endothelial cells, this giving rise to weakened points in the vessel wall. Consequently, the adherent leukocytes, by virtue of their active ameboid movement, very soon begin to *emigrate* through the vessel walls in great numbers. The cell sends a minute process through a softened cement line of the veins and capillaries (not the arteries), a knob appears on the outside of the vessel, and the remainder of the cell follows, until finally its entire body is found in the surrounding tissues outside the vessel wall. This cell dies and forms a pus-cell, remains attached to the vessel and organizes into a living tissue cell, or it may wander off in the lymph-spaces. This phenomenon of emigration, it must be understood, is not a passive infiltration, but an active process accomplished by the ameboid movement of the cell, and made possible by the softening of the cement substance.

If the inflammatory process be very severe, or if large numbers of leukocytes have emigrated, the cement lines become so permeable that the red blood-cells are permitted to pass through in rapid succession, under the influence of pressure. This process is called *diapedesis.* Accompanying and usually preceding the emigration of white and red blood-cells a fluid exudate is poured out into the surrounding tissues, which represents the serum of the blood, modified in some unknown way as it passes through the vessel walls. The process is not one of filtration, but a disordered secretory function of the endothelium, brought about by the change in the vessel wall. This fluid contains a high percentage of albumin, and may also contain the fibrin ferment necessary to cause a coagulation of the exudate with the separation of fibrin. This will account for the serum and fibrin of inflammatory conditions.

The fate of the various materials which have exuded in this way from the blood-vessels must necessarily be different in the various tissues of the body. In the arm, for example, the serum, fibrin, and red and white blood-cells will be poured out into the connective tissue in large amounts, render the structures edematous and boggy, and produce a condition called *cellulitis.* Serum secreted on a serous surface will form large collections of fluid in the body cavities, as in pleurisy with *effusion.* If the exudation is cast off on a mucous surface we have a *catarrhal* inflammation, as in pneumonia and bronchitis. These exudates on a mucous surface may coagulate and form a *croupous exudate.*

It remains to account for the small round-cells which are found so abundantly in all inflamed tissues, because they constitute a very important feature in the lesion. They are, undoubtedly, entirely composed of the white blood-cells exuded from the vessels. The connective-tissue cells among which the exudate takes place proliferate a little later, but even then the newly-formed cells have not the features of lymphocytes. The leukocytes which are exuded may, in part, return unchanged to the blood by way of the lymph-channels; others take on very rapid proliferation, resulting in a great increase of cells in the tissues to form the small round-cells

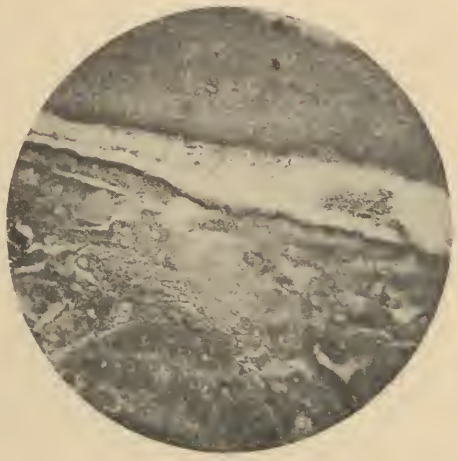

FIG. 1.—CROUPOUS INFLAMMATION.

The lower portion of the diagram shows the mucosa of the trachea infiltrated by pus-cells and its vessels are dilated. Above and separated from the mucosa by a clear space is the false membrane, made up of pus-cells, epithelial cells, and fibrin.

spoken of above; while still others die and form pus-cells, which either collect to produce abscesses, are thrown off as an exudate or false membrane on free surfaces, or disintegrate and are absorbed.

Such an exudate, collected in the tissues, may occlude the arteries and produce necrosis and disintegration. Much more frequently, however, the circulation is preserved, the exudate begins to soften with the recession of the inflammatory condition, and is either disintegrated and absorbed or cast off on free mucous or serous surfaces. The serum element is rapidly absorbed by the lymph-vessels, but when contained in serous cavities may remain for a long time. The red blood-cells lose their color, disintegrate, and

are absorbed. The fibrin is either present in such amount as to occlude the arteries and cause death of the tissues, or it softens and disappears, or may become organized into new connective tissue.

Any one of the products of exudation may predominate and give rise to *serous*, *fibrinous*, *purulent*, or *sanguineous* inflammations. Serous exudation into tissues causes *edema;* on a mucous surface, *catarrhal inflammation.* When plastic lymph is thrown out, we speak of the exudate as *fibrinous.* This exudate is more or less solid in consistency, and forms patches adherent to the surfaces from which it springs. When a fibrinous exudate is confined to a

FIG. 2.—PURULENT MYOSITIS.
Pus-cells are found in abundance between the muscle-fibers.

serous cavity, there is usually more or less serous fluid present also, making the exudate *serofibrinous.*

A *hemorrhagic exudate* most often occurs as a result of intense inflammatory reaction, and is usually associated with the formation of fibrin, as in pneumonia. An exudate made up mainly of leukocytes is found in the mucous membrane as a *purulent catarrh*, in the cavities of the body as *empyema*, and in connective tissue as *purulent infiltration*, *abscess formation*, *ulcers*, etc. The exciting cause in any purulent inflammation is usually some one of the pus-forming bacteria.

II. The term **necrotic inflammation** is one used to cover a

number of conditions where, added to the preceding phenomena of simple inflammation, there is also a death of tissue present as the characteristic element.

(1) *Purulent* or *suppurative* inflammation is the most common, and perhaps the most important, of this variety. It is an inflammation similar in every way to the simple inflammation just described, except that the exudate consists mainly of polynuclear leukocytes in excessive amounts, and these cells are called *pus-cells.* These pus-cells may infiltrate the tissues diffusely and be found crowded between the elements of the tissue, as, for instance, between the muscle-fibers in an interstitial myositis, or they will be found closely packed in the tissues, to complete obliteration of the former structures. When there is such a collection of pus-cells, the tissues involved soon begin to look yellowish-white, then break down, liquefy, and form *abscess cavities* filled by pus-cells and broken-down tissue fragments. When the pus-cells collect in body cavities, as the pleura, joints, antrum of Highmore, etc., they form purulent effusions, or *empyemata.* Such suppurative inflammation, occurring in the skin or mucous membrane, forms *ulcers;* or, if the exudate be cast off on a mucous surface, it constitutes a *purulent catarrh.* When there is great transudation of serum along with the pus, it constitutes a *seropurulent exudate*, and if this exudate invades an extensive area, a *phlegmon* is produced. Likewise, on serous surfaces we may have a *fibrinopurulent* exudate.

The suppurative inflammations are usually caused by the presence in the tissues of the pus-forming bacteria, and most frequently by the Staphylococcus pyogenes aureus and Streptococcus pyogenes. Chemical substances, however, such as turpentine, nitrate of silver, mercury, croton oil, sterilized cultures of bacteria, etc., may produce suppuration when introduced into the tissues, yet such an inflammation has none of the virulent characteristics of the bacterial form.

(2) The next variety of necrotic inflammation is one where *suppuration does not* follow the tissue necrosis, but the areas of dead tissue, which are of definite size, remain unchanged for a length of time, until they are either cast off in bulk or are slowly absorbed. Such a condition is caused by the action of caustic chemical substances, arrest of the blood-supply to a part, high and low temperatures, and bacterial infection, such as that produced by the bacteria of typhoid fever, diphtheria, and dysentery. The diph-

theric form of inflammation occurs on mucous membranes as a coagulation necrosis, affecting both the exudate and the inflamed and indurated tissues.

(3) The remaining type of necrotic inflammation is one in which the necrosis appears only after new inflammatory tissue has formed and existed for a long time, as in tuberculosis. Here the necrosis is called *cascation.*

III. **Productive Inflammation.**—According to Dr. Delafield, we may have: (1) A simple, acute, productive inflammation where there is *no* exudation of serum, fibrin, or pus, but the products of inflammation are entirely represented by new connective-tissue

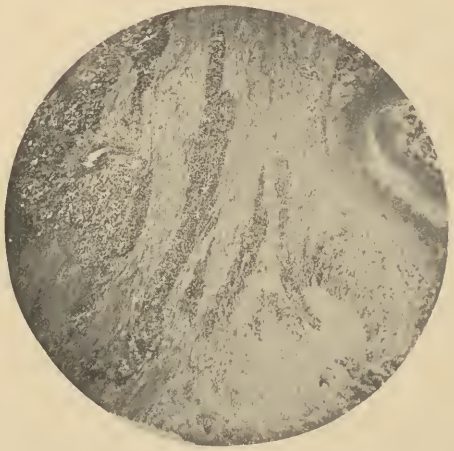

FIG. 3.—INTERSTITIAL INFLAMMATION IN A LYMPH-NODE.
New connective tissue has formed through the node, until only a few islands of cells are left.

cells, formed from the old. (2) A productive inflammation *with* exudation. The usual products of exudation are present, but there is also, from the start, a production of new tissue, which is first composed mainly of cells, but later develops fibers and vessels. This gives rise to the subacute and chronic types of inflammation. The exudate may be, and usually is, absorbed, but the new tissue is permanent, and its organization is usually progressive. The new tissue affects mainly the stroma. (3) There may be a chronic productive inflammation, with or without exudation, but with the formation of round-cell tissue, granulation tissue, or connective tissue. This is the type often called *interstitial inflammation.*

Such productive inflammations are very slow in their progress, and tend to continue indefinitely. They are caused by slight but long-continued irritation, as from inhalation into the lungs of coal-dust; by long-continued action of the poisons of syphilis, gout, rheumatism, and alcohol; and by bacteria, in the case of tuberculosis and other of the infective granulomata. Just the relationship between this type of inflammation and a simple fibrosis occurring in the repair after a simple inflammation, is not yet understood.

## HEALING.

The process of **healing** or **repair** after simple inflammation or destruction of tissues must necessarily vary much: (1) With the nature of the causative agent, whether applied momentarily, as in trauma, or during a considerable period, as in bacterial action. (2) Again, it must be very different where there is slight loss of tissue, and where healing takes place by granulation and cicatrization, after extensive loss of substance. No matter what the organ or the character of the tissue destroyed, every extensive loss of substance is replaced by connective tissue. Repair is best understood by studying an open wound of soft parts on the body surface, healing by granulation, and not infected by bacteria.

After hemorrhage has ceased, the blood-vessels dilate, the tissues are swollen, serum, fibrin, and white blood-cells exude upon the surface and into the tissues, and fragments of tissue necrose. On the second or third day the surface is covered with small red papules, called granulations, and is bathed in an exudate rich in albumin and dead leukocytes or pus-cells. New blood-vessels begin to form at once on the surface of such a wound, by a growth of buds of solid protoplasm from the cells of the walls of old blood-vessels. These buds lengthen out, and are channeled by the blood-pressure from within the vessels, with a simultaneous appearance in the protoplasm of nuclei to form endothelial cells. These new vessels contain circulating blood, form abundant anastomoses by giving off sprouts themselves, and, by their projection above the normal surface form the papillæ which characterize a granulating surface. New connective tissue, of an embryonal type, has meanwhile been forming around these new blood-vessels, as a result of proliferation both from the fixed connective-tissue cells and from the walls of

the vessels. This tissue is made up of small round-cells, hypertrophied connective-tissue cells, and round and polynuclear leukocytes closely packed together, the only partition being a fluid intercellular substance. From the small round-cells are very soon developed certain large polynuclear cells, with large, oval, clear nuclei, which stain less deeply than those of the other cells. These are the formative connective-tissue cells, and are called **epithelioid** cells, from their resemblance to epithelium. They increase rapidly in number, until they predominate over the round-cells, and are packed more or less closely together. Certain of these cells, and especially those cells representing hypertrophied connective-tissue

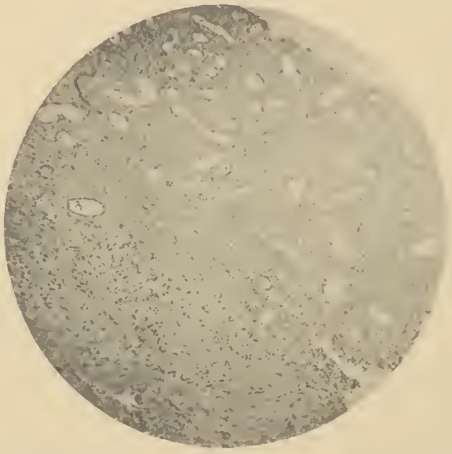

FIG. 4.—GRANULATION TISSUE FROM THE INNER WALL OF A GRANULATING OVARIAN CYST.

cells, become much enlarged, and spindle-shaped, or branched, to form the **fibroblasts**, which seem to be especially concerned in the formation of connective-tissue fibers, and which remain as fixed connective-tissue cells. At a certain stage in their development these fibers appear in the intercellular substance as branches from the cells, or as a formation of fibrillæ alongside of them. The round-cells not undergoing these changes seem to die, and are thrown off on the surface of the wound as pus-cells. With the formation of connective-tissue fibers the cells become progressively fewer in number, until the entire tissue is made up of fibers with a few flattened fusiform or branched cells, and we have the condition known as a *cicatrix*. In other words, *healing is complete*.

When the wound is in the skin, the formation of granulation tissue continues until the epithelium, by progressive extension over the granulation surface, has covered the entire surface of the latter.

The difference between **healing by first intention,** or primary union, and *healing by granulation* in an open wound, although of great importance to the surgeon, is one entirely of degree. In an incised wound, where the parts are drawn accurately into apposition, there is slight necrosis of tissue, and the area into which exudation may take place is of such paper thinness that granula-

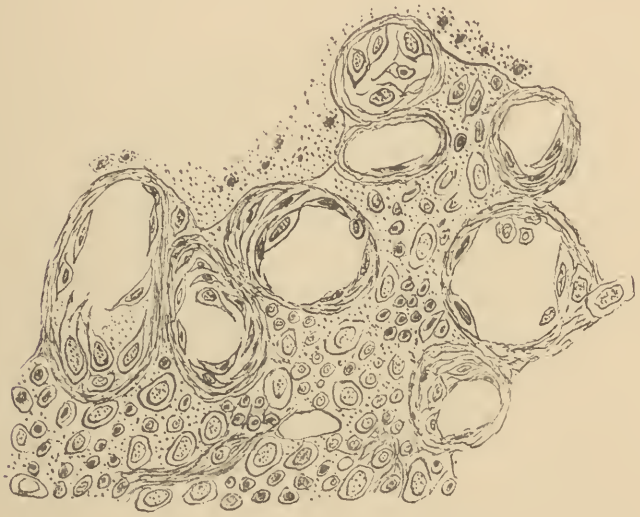

FIG. 5.—EXUBERANT GRANULATION TISSUE.

Blood-vessels with rapidly proliferating endothelium predominate and project above the surface. Many types of cells are found in the granulation tissue.

tions can very quickly fill it. Every stage in the process of healing is identical with that described above, except that the amount of new tissue formed is very much less, and the resulting cicatrix is a mere line.

When *fibrin* forms on an inflamed serous surface, granulation tissue very soon forms beneath it. The fibrin is penetrated from below by vessel sprouts, new tissue cells form, and the mass of fibrin is soon transformed into connective tissue. This results either in a thickening of the serous coat, or, when two serous surfaces are opposed, in adhesions by connective tissue bands.

*Coagulated exudates* in the lung may undergo the same organization, and produce the organized or indurated pneumonia. *Thrombi* and *necrotic areas* that can not be cast off externally, organize in the same way. Likewise, in *abscess* cavities and empyemata, the tissues surrounding the necrosed area begin to heal, form granulation tissue, and, with the absorption or evacuation of the pus, the cavity will be filled by new granulation tissue and eventually cicatrize.

In some granulation surfaces the tissue growth does not keep pace with the formation of new blood-vessels, and the surface is one mass of thin-walled capillaries, projecting high above the surface. Flabby, pale granulations are formed and the wound

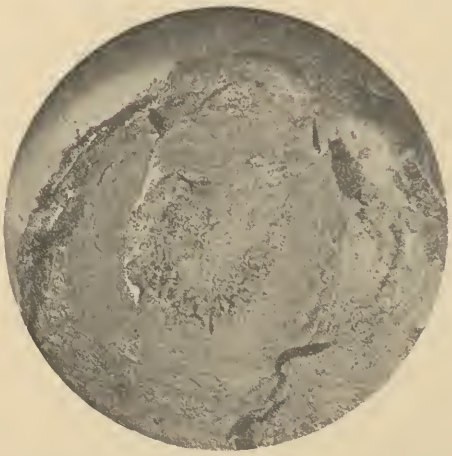

FIG. 6.—ORGANIZED THROMBUS IN ANTERIOR TIBIAL ARTERY.

Man aged thirty-five, gave symptoms of slow but progressive gangrene of toes and foot. The lumen of the vessel is filled by a typical granulation tissue, rich in blood-vessels.

does not heal. These are called *exuberant granulations*, or "proud flesh." Certain chronic granulations form spongy, tumor-like growths, and are called *fungous granulations* or **infectious granulomata.** These are caused by a specific infection, as by the germs of tuberculosis, syphilis, leprosy, glanders and farcy, rhinoscleroma, and actinomycosis.

Inflammation may pursue a **chronic** course (1) because the tissues may be unable to recover immediately from great loss of substance, and regeneration must go on slowly. (2) There may be present a continued irritation, as is seen in the progressive connective-tissue thickening in the lungs as the result of the inhalation of

coal- or stone-dust, or as is seen in the formation of condylomata on parts constantly bathed with acrid discharges. (3) Chronic congestion plays an important part and acts, doubtless, by disturbing the nutrition of the parts. This is illustrated by the varicose ulcer of the leg or by chronic inflammation of any one of the viscera following a chronic congestion. (4) Infection by bacteria or molds, as is seen in gonorrheal and tubercular inflammation of mucous membranes. (5) Chronic intoxications which seem to affect mainly the liver and kidneys, as is seen in the poisoning of syphilis, alcohol, lead, etc.

### Healing of Bone Fractures.

The healing of bone varies from that taking place in soft tissues, alone in the fact that the lime salts are present and the cicatrix is bone. With a fracture of a bone the periosteum and surrounding tissues are lacerated and blood is poured out between the ends of the bone and forms a fusiform tumor around the seat of injury. A simple exudative inflammation follows, with an infiltration of the tissues by leukocytes and serum; the blood is absorbed, and in a very few days, with a subsidence of the inflammation, a soft granulation tissue is formed. This granulation tissue is formed (1) around the bone, and extending for some distance above and below the line of fracture, to form a spindle-shaped tumor, which will later become the *ensheathing callus;* (2) it replaces the medulla for some distance up and down the canal, to form, later, the *internal callus;* and (3) it forms between the ends of the fragments, receiving its new capillary buds from the vessels of the Haversian canals, and gives place to the *intermediary callus.* The ends of the bone, meanwhile, have undergone a rarefying osteitis and become soft and porous, and the granulation tissue entirely changes into cartilage or into firm connective tissue, called *osteoid tissue.* Ossification begins around the vessels in the angle between the torn up periosteum and the bone. Irregular branching lamellæ of bone form through the callus under the action of the *osteoblasts*, Haversian canals continuous with those of the bone form between the lamellæ, and the ends of the fracture are surrounded by a mass of spongy bone, which lasts until healing is complete. Meanwhile, the medulla has been undergoing the same changes, and is also filled by newly-formed bone. The soft, rarefied ends of the bone show an enlargement of the Haversian canals, and are united by a mass of spongy bone.

When union is completed an absorption of the callus takes place. This begins first in the medullary canal, which again becomes per-

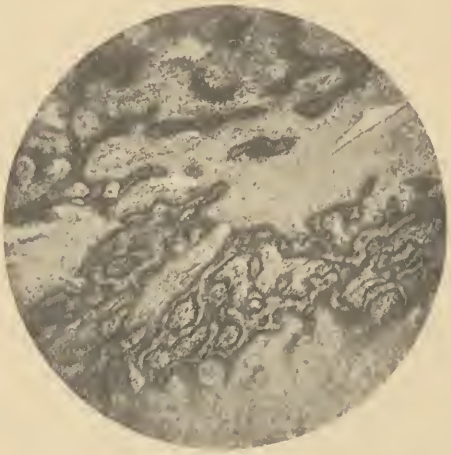

FIG. 7.—HEALING OF BONE FRACTURE.
Laminæ of new bone are forming through the granulation or osteoid tissue.

vious, and there is a gradual disappearance of all excess of bone and a return to normal. With imperfect apposition of the ends of

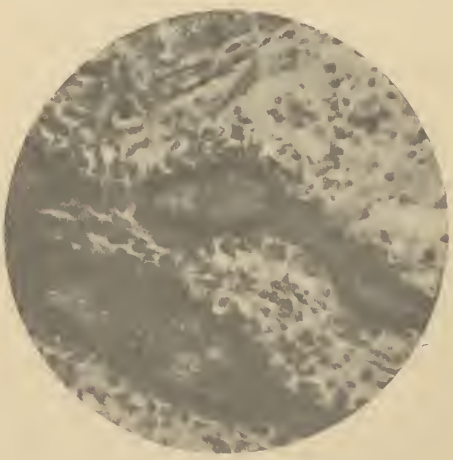

FIG. 8.—HEALING OF BONE FRACTURE.
Two laminæ of new bone are represented with osteoblasts along their borders.

a fracture, angular deformity, etc., a large mass of callus remains permanently.

### Repair of Nerves.

After the division of a nerve there is a degeneration in the entire peripheral portion, and also, to a limited degree, in the central end of the nerve, dependent undoubtedly upon the separation from their neurons. The medullary sheaths break up into droplets, which degenerate, become fatty, and after a time are absorbed, and the axis-cylinder shares in the destruction and complete absorption. The neurilemma and its nuclei, however, are preserved, and it is from these *nuclei* that regeneration takes place. By the seventh day an active proliferation sets up in these nuclei, and they are found free in the cavity of the internodal space; yet protoplasm soon collects around them, and a fusion of such cells forms a solid rod of protoplasm, or an *embryonic nerve-fiber*. A new nerve-sheath is formed from the outer layers of the protoplasmic cord, and healing is complete. These changes of regeneration go on, provided the divided nerve-ends are reunited by suture or otherwise; but if they remain apart, the proliferation of nuclei is slow at the beginning, and finally ceases without forming the embryonic fiber. It will be observed that this regeneration is entirely from pre-existing nerve tissue, and not from the connective tissue of the perineurium and endoneurium.

## TUBERCULAR INFLAMMATION.

Tubercular inflammation is a specific inflammatory process caused by the presence and growth of the *tubercle bacillus*. This is a disease affecting both man and animals, and consequently in any inhabited region immense numbers of bacilli are thrown off in the sputum and excreta and are spread broadcast. These germs retain all of their virulence, even when dried, and are ready to set up a local tubercular inflammation at their point of lodgment whenever they gain entrance into the body. They gain entrance most frequently, of course, to those places which are accessible from without, as, for instance, the lungs, intestines, and skin; yet there are many cases where the first indication of lesion is in some deep-lying tissue, as the testicles, lymph-nodes, bones, or joints, and we must infer that the germs gain entrance into the blood- and lymph-vessels through some imperceptible abrasion on the surface of the body and leave no evidence of infection at their point of entrance.

The tubercle bacillus sets up in the tissues a proliferation of the fixed connective-tissue cells, and an exudation of white blood-cells

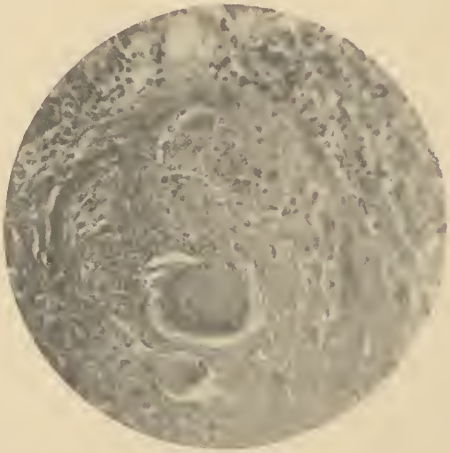

FIG. 9.—TYPICAL MILIARY TUBERCLE, FROM A LYMPH-NODE IN A CHILD.
A giant-cell, epithelioid cells, and small round-cells are present.

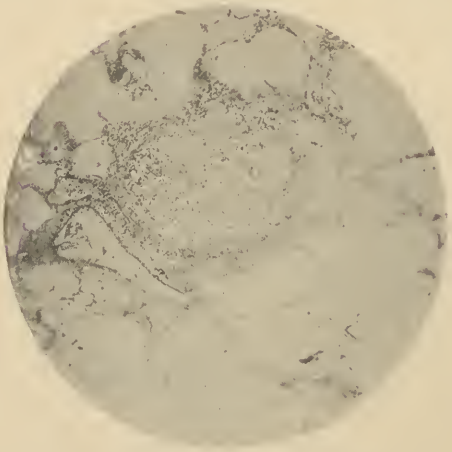

FIG. 10.—VESSEL INFECTION IN TUBERCULOSIS.
The clear carrot-shaped space to the left represents a blood-vessel, in the wall of which tubercle bacilli have lodged to form the miliary tubercle.

from the vessels with the formation of circumscribed nodules of cells, which remain devoid of blood-vessels and after a time necrose. The first effect of the presence of the tubercle bacillus in the

tissues is a stimulation of karyomitotic proliferation in the fixed connective-tissue cells, with the development of large mononuclear cells resembling epithelium and called *epithelioid* cells. At the same time one or more giant-cells, containing many large oval nuclei, are usually formed in the rapid proliferative process. These cells form more or less definitely circumscribed spheroidal masses around the bacilli, and are called **tubercles** or **miliary tubercles.** The reticulum of the normal tissues is pushed apart by the new forming cells, until a fine network alone separates the cells, or this reticulum of the tubercle is a new formation going on with the development of the cells. New blood-vessels do not form in this

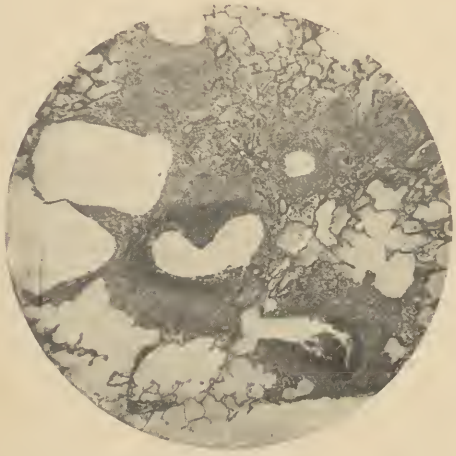

FIG. 11.—BRONCHIAL INFECTION IN TUBERCULOSIS.
A bronchus is shown in the center of the field, the walls of which are replaced by tubercle tissue.

new tissue, and the old vessels are obliterated as the process invades them. Besides this obliteration set up in the blood-vessels of the tissues invaded by the tubercular process, there is a further alteration in the surrounding vessels, which results in an exudation, sooner or later, of large numbers of *leukocytes*, which latter also enter into the formation of the tubercle. The exudation of white blood-cells may be very limited and the miliary tubercle consist mainly of epithelioid and giant-cells with a few small round-cells; or, on the other hand, it may take place very early, and in this case the small round-cells will characterize the tubercle throughout, and the large epithelioid cells will be either entirely absent or appear

only at a later stage. Such a completely formed tubercle appears in the tissues as a small, circumscribed, translucent collection of cells about the size of a millet-seed. When it has reached this development, however, retrograde changes take place by which the cells in the center of the tubercle undergo that form of necrosis known as cheesy degeneration, and the nodule becomes a cheesy mass surrounded by a zone of cells. The tubercle now has an opaque yellowish-white appearance. The subsequent history of such miliary tubercles varies considerably: (*a*) Secondary infection by the pus-forming bacteria may take place, with rapid breaking down and abscess formation; (*b*) fibrous tissue may form around the miliary tubercle and lead to its complete obliteration and cure; or (*c*) new tubercles may form about the primary focus through the lymph-channels, thus leading to extension of the disease and its diffusion throughout the organ.

It is evident that there may be many types of miliary tubercles:

1. The tubercles may be wholly made up of small round-cells.
2. They may be entirely made up of large epithelioid cells.
3. There may be both epithelioid and round-cells uniformly intermingled, or the small round-cells may be arranged in a zone around a central collection of epithelioid cells.
4. Giant-cells may be, and usually are, mixed with the preceding types.
5. Caseation takes place in the center of any one of these forms.

**Diffuse Tubercular Inflammation.**—When there is a very extensive infection by tubercle bacilli, the miliary tubercles will be very numerous. These extend, as has been explained, by the formation of other tubercles about the primary focus through the agency of the lymph-channels, and great numbers of tubercles run together in this way to form extensive areas of tubercle tissue in more or less advanced caseation. Such areas are found in the lungs, brain, serous membranes, lymph-nodes, kidneys, prostate, testicles, etc.

*Another type* of diffuse tubercular inflammation is that which we find in an acute phthisis, where the predominant element is a severe inflammatory reaction to a very virulent infection, with the pouring out of large quantities of the products of simple inflammation. These products undergo very rapid caseation before the typical organized tissue of small round-cells has had time to form.

**Disseminated tuberculosis** is the name sometimes given to

that form of the disease where one organ after another is infected from a primary focus. Thus, with a primary tuberculosis of the lung there may be a secondary tuberculosis of the liver alone, of the kidney alone, of the meninges, or of several organs simultaneously, or of one organ after another. This dissemination occurs, of course, by a breaking through of the tubercle bacilli into the blood-current, whence they are carried to distant parts.

## LUPUS.

Tuberculosis may occur in the skin as a result of direct infection, or about sinuses leading to areas of tubercular inflammation, but the typical skin tuberculosis is **lupus**. This is characterized by the appearance of multiple small, reddish-brown nodules of granulation tissue in the skin of the face, and, much less frequently, in the mucous membrane of the mouth, conjunctiva, vulva, and vagina. These nodules may run together, forming a more or less extensive area of infiltration; the epithelium over them is increased excessively in amount, and the nodules break down and ulcerate, or they degenerate, absorb, and leave a white scar, without ulceration. The disease begins between the age of two years and puberty, and its course is always chronic. Microscopically, the nodules consist of small round-cells of granulation tissue, epithelioid cells, and often giant-cells. These cells are supported by a well-developed reticulum, and the nodule differs from a miliary tubercle of other parts in being rich in blood-vessels. The tubercle bacillus has been found constantly present in small numbers.

The course of tuberculosis is usually chronic. Ulcers form if the broken-down nodule is on the skin, or cavities and systems of cavities, lined by a pyogenic membrane, form when the lesion is deep in the tissues. Extension may be by the lymph-channels, or foci may break into an artery, when the bacilli will be carried to distant parts, and form metastatic growths of tubercle tissue. It has been found by Prudden that dead tubercle bacilli set up the same proliferation of the tissues, with the formation of miliary tubercles identical in structure with those caused by living germs; but coagulation necrosis and systemic intoxication are absent.

## SYPHILITIC INFLAMMATION.

Syphilitic inflammation occurs only in man. It bears a close analogy to tubercular inflammation, and is caused by a bacillus described by Lustgarten, which also resembles very much in its appearance the tubercle bacillus. The bacillus of syphilis, however, produces the greatest variety of inflammatory reactions, ranging from a simple hyperemia to extensive tumor formation or connective-tissue growth. Infection takes place by direct or indirect transfer of the virus from one person to another, or it may be inherited.

The *primary focus of infection*, or the **hard chancre**, or Hun-

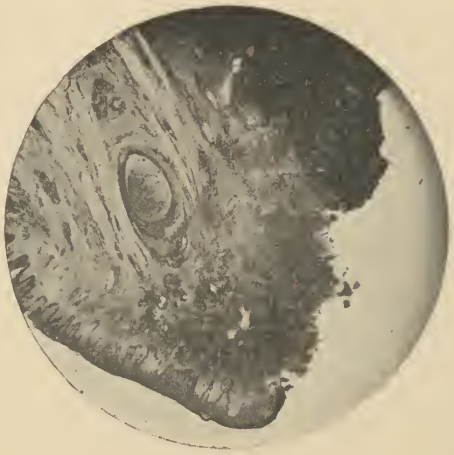

FIG. 12.—CHANCRE OF THE LIP.

The skin surface is shown on the left and the surface of the ulcer on the right, with a round-cell infiltration deep into the tissues.

terian induration, is a circumscribed area of dense, small round-cell infiltration in which are found a few epithelioid, and, somewhat later in its development, giant-cells. The area is hard, and remains fibrous or breaks down, ulcerates, and heals by cicatrization. The **secondary** lesions consist of an inflammation of the lymph-nodes, skin, and mucous membranes. In these lesions there is a round-cell infiltration, associated with a hyperplasia of the fixed tissue cells. In the skin the papillæ especially are infiltrated with cells and fluid exudates, the epithelium is increased in amount, is infiltrated by round-cells, and is gelatinous. The syphilitic lesions occurring in the internal organs and belonging to the tertiary stage

are usually called **gummata.** These appear in the periosteum, brain, liver, spleen, testicles, etc., as grayish-white or grayish-red firm masses, with a cheesy center and a translucent outer zone. They are usually rich in small round-cells, and seem to be true granulation tissue, with new but poorly-formed blood-vessels. These gummata undergo coagulation necrosis or cheesy degeneration, and their peripheral portions merge into connective tissue, which surrounds the caseous masses. This connective tissue radiates in rays into the surrounding tissues.

Syphilitic **endarteritis** constitutes an important lesion of syphilitic inflammation. The changes which take place, especially in the

FIG. 13.—VENEREAL WART FROM THE VULVA.
There is a slender branching stalk of fibrous tissue, covered by thick layers of epithelial cells.

smaller arteries and veins of the brain, consist in a growth of new tissue, resembling granulation tissue, beneath the endothelium which leads to a gradual narrowing of the lumen. This tissue later becomes more fibrous; furthermore, the endothelium becomes altered and the blood coagulates to form *thrombi*, with resulting cerebral softening.

## LEPROSY.

Leprosy is an infectious disease which is common in India and the hot countries, and there are isolated cases in other regions. It

is caused by the presence of the lepra bacillus, and is characterized by the formation in the skin of exposed parts (rarely in subcutaneous tissue, viscera, etc.) of nodules or masses of granulation tissue, made up of small spheroidal cells, large round-cells, and branching cells supported by a stroma. These nodules are minute, or one inch in diameter; they push aside, are intermingled with, or cause an hypertrophy or an atrophy in the tissues invaded. The nodules remain a long time, or ulcerate, or form white cicatrices without ulceration. Certain nerve lesions are secondary to the disease.

## GLANDERS.

This is an infectious disease affecting the nasal mucous membrane of the horse, which is caused by the Bacillus mallei, and may be communicated to man. Occurring in the skin of the horse, it is called **farcy**. The seat of infection in man is usually the skin, but it may be found in the mucous membrane, lungs, kidneys, testes, spleen, and liver. Small round and epithelioid cells (no giant-cells) infiltrate the tissues diffusely or are collected in circumscribed masses, which appear as small white foci, much resembling miliary tubercles, or as large-sized nodules. These soon break down, to form abscesses or ulcers when near the surface, and the skin will be covered with a pustular eruption, and the deeper structures contain abscesses of varying size. The nodules may pursue a very chronic course without abscess formation, and such persistent masses are differentiated from miliary tubercles by the absence of cheesy degeneration and giant-cells in the former. The bacillus is abundant in fresh nodules, but found with difficulty in the degenerated nodule.

## RHINOSCLEROMA.

This is a chronic inflammation of the mucous membrane of the nose, pharynx, and larynx, caused by the Bacillus rhinoscleromatis, and characterized by the formation of granulation tissue in nodules and masses, which soon become dense and cicatricial.

## ACTINOMYCOSIS.

Actinomycosis (*ray fungus*) is an infectious disease, caused by the actinomyces, which is a micro-organism belonging undoubtedly among the bacteria. The germ develops to form radiating filaments with club ends, hence the name "ray fungus." In cattle it constitutes "lumpy jaw," whereas in man a granulation tissue is formed around the fungous mass, making a tumor formation, which is characterized by early necrosis and sloughing, with abscess formation. Such tissue is found in the lungs, skin, lymph-nodes, gastro-intestinal canal, etc. The fungus is diagnostic, and appears as yellow masses, the size of a millet-seed, scattered through the new tissue and contained in the pus.

## DEGENERATIONS.

In the tissues of the body are certain retrograde changes in the structure of the tissues, dependent upon general or local causes, and leading to a diminution in the size of any given organ, the destruction of its element, and an impairment or total loss of its function. There are many types of degeneration, each one of which will be taken up in order.

### Necrosis.

Necrosis is a local death of tissue cells, with consequent cessation in their function. It is caused by lack of nutrition, diminished oxygen supply, mechanical injury, high temperature, chemical agents, poisons, and trophic nerve disturbance. While these may be given as exciting causes, the effect produced, however, by their action, seems largely dependent upon the condition of the tissue at the time when such causes act. The cells may gradually lose their nuclei, break down, soften, and form a fluid, degenerated mass, or they will be swollen and granular, disintegrate, become pigmented by broken-down blood-cells, and contain bacteria. The surrounding tissues are inflamed. Complete regeneration may take place in

the degenerated areas, or cicatrization, calcification, or cyst formation may follow.

### Coagulation Necrosis.

In coagulation necrosis the cells become transparent, lose their nuclei, are shrunken and distorted. This is due either to certain chemical elements present (ferments or bacterial products), producing coagulation of the albuminous constituents of both exudate and cell, or to normal body fluids flowing over the necrotic area.

### Cheesy Degeneration.

Cheesy degeneration is a term used to designate that type of degeneration in which the cells lose their nuclei, become granular, fatty, or homogeneous, and run together to form masses resembling hard or soft cheese. This mass is made up of fatty and albuminous debris, of granules and indistinct fragments of tissue, but no cell outlines. It occurs in tubercular and syphilitic inflammation.

### Liquefaction Necrosis.

In liquefaction necrosis the degenerated tissues are infiltrated by liquids, break down, and become fluid.

### Gangrene.

Gangrene is death of tissue large enough to be seen with the naked eye. **Dry gangrene** results from a gradual stoppage of arterial supply, with unimpaired venous return, while in **moist gangrene** there is a sudden stoppage of arterial supply or of venous return, with decomposition and putrefaction of the necrotic tissues.

### Hypoplasia.

Hypoplasia is a defective development of the entire body, of certain organs, or parts of organs.

### Atrophy.

Atrophy is a diminution in the size of an organ, dependent upon a decrease in the size or entire disappearance of the structural elements composing it. It may be a *simple atrophy*, with no change in the structure except the diminution in size of the elements; or it may be a *degenerative atrophy*, where the cells become granular or

fatty, lose their nucleus, swell up, then degenerate, liquefy, and disappear. Atrophy may be senile, from impaired nutrition, inflammation, pressure, disease, or it may be neuropathic.

### Cloudy Swelling.

Cloudy swelling, or parenchymatous degeneration, is a degenerative process taking place in the parenchyma cells of organs, and is found in the acute infectious diseases, fever, burns, and poisoning from phosphorus, arsenic, and acids. The cells swell up, become cloudy, and filled with small albuminous granules, so that the normal structure and form of the cell is lost. It is a beginning disorganization of the cell protoplasm, and the cell may go on to complete disintegration or return to its normal condition. Such an organ appears gray and cloudy, anemic, and, in the more advanced cases, is doughy in consistency, and looks as though it had been boiled.

### Fatty Degeneration.

Fatty degeneration is a degenerative process in which minute fat droplets are formed in the cell at the expense of its protoplasm. The fat droplets are very small, as a rule, but may coalesce to form larger drops. In far advanced cases the cells may be broken up into a cell detritus, composed of granules, fat droplets, and sometimes cholesterin crystals. It is a process which is undoubtedly dependent upon a lessened food and oxygen supply to the organ, together with a lowered vitality on the part of the cell itself. Thus, in anemia from hemorrhage, or in certain diseases associated with profound anemia, and with many poisons, we are apt to find this condition.

### Fatty Infiltration.

Fatty infiltration is a condition where the fat is formed outside the cell body and deposited in its *protoplasm*. The fat droplets are much larger than in fatty degeneration, as a rule, and the condition is less serious.

### Mucous Degeneration.

Mucous or mucoid degeneration has its analogue in the normal physiological production of mucus in the epithelial cells of the mucous membrane and glands. Here the mucous cell becomes transparent, swells up, loses its granules, and discharges its mucus. Pathologically, the protoplasm of the epithelial cells and connective-tissue group may degenerate into this type of tissue, and take on

a translucent, gelatinous, and swollen appearance from conversion into mucus. In connective tissue the degeneration may be confined to the intercellular substance, and the cells suffer only secondarily, by pressure and lack of nutrition. The gelatinous substance found in such degeneration is of complex chemical composition, with mucin and pseudomucin as the important constituents, and it is precipitated by acetic acid. It occurs in mucous membrane, tumors, cartilage, and bone.

### Colloid Degeneration.

Colloid degeneration is a change closely related to the preceding form, but it takes place in the cell protoplasm alone, and not in the connective tissue. The colloid material collects in droplets of varying size, and may cause complete degeneration of the cell. It has a homogeneous appearance similar to mucus, but is usually firmer and more consistent. It does not swell or dissolve in water and is not precipitated by acetic acid or alcohol. Colloid material normally exists in the thyroid gland, and this is the seat of its most frequent pathological formation. It also occurs in the renal tubules, the gastric mucous membrane, and the pituitary body of the brain.

### Amyloid Degeneration.

Amyloid, *waxy*, or *lardaceous* degeneration is a peculiar degenerative process, where a firm, clear, albuminous substance, called amyloid material, is deposited in the walls of the blood-vessels and in the connective tissue of various organs of the body. The spleen, liver, kidney, intestinal tract, suprarenal capsules, lymph-nodes, and connective tissue are most frequently affected, and in the order named. The organ increases in size, becomes firm, and presents, on cut surface, clear, waxy areas, which may be seen with the naked eye when the process is extensive. Hence the name "waxy liver" and "sago spleen." When fresh tissue is treated with a solution of iodin and potassium iodid (Lugol's solution), the amyloid areas are brought out in mahogany-red color. When sections are stained deeply in a one per cent. aqueous solution of methyl-violet and mounted in glycerin, the amyloid material is stained a rose-red color, while the remaining tissues are stained deep-blue. Amyloid material is a firm, translucent, or glassy albuminous material, which is clearly deposited in the walls of the arteries and capillaries and between the fibers of connective tissue.

The wall of the blood-vessel appears as a thickened ring with a progressively narrowing lumen. The parenchyma cells of the organ are not invaded, and suffer only from pressure when the deposit is great. It occurs in cachectic conditions, especially with suppuration and bone disease, tuberculosis, syphilis, dysentery, leukemia, etc. The impaired nutrition of the cells in such conditions seems to favor the formation of amyloid material from certain materials contained in the blood. The name amyloid was given because of a supposed resemblance to starch, but it is now proven to be a nitrogenous and not a carbohydrate substance.

**Corpora amylacea** are amyloid concretions found in the central nervous system and prostate gland. They occur both normally and under pathological conditions. These bodies, however, do not appear to belong to the type of progressive amyloid degeneration, although they do give the amyloid reactions. Some appear to be formed from the albumin of the affected tissues, while others are made up of degenerated epithelium.

## Hyaline Degeneration.

Hyaline degeneration is a process closely allied to the preceding form, and occurring in the walls of the smaller blood-vessels and, occasionally, in connective tissue. It does not give the amyloid reaction. Hyaline material is a transparent, glassy substance, staining deeply with eosin and deposited immediately beneath the endothelium of the blood-vessels, thus rapidly diminishing their lumen, often to complete obliteration.

*Seat of Occurrence.*—It occurs in the brain, lymph-nodes, ovaries, kidneys, eyes, etc., as a result of tubercular or syphilitic disease. A form of this degeneration, belonging to old age and occurring in the valves of the heart, blood-vessels, thyroid gland, and the stroma of tissues, has been called by Virchow *sclerosis*.

## Calcification.

Calcification is a condition where granules of the phosphate and carbonate of calcium are deposited both in the cells and stroma of tissues. The cause is undoubtedly a preceding degenerative change of the tissues, leading to an attraction of lime salts to them.

### Pigmentation.

Pigment exists normally in the body in the rete Malpighii, the choroid, ganglion cells, red blood-cells, etc. Pigment may be deposited pathologically in the cells and stroma of many different tissues as yellow, brown, black, or red granules, but its origin and formation are no better understood than that taking place in normal tissues. Pigment formed by the breaking down of red blood-cells may be in the form of red plates and needle crystals of *hematoidin*, or it may be the yellow or brown iron-containing pigment *hemosiderin*, which gives rise to the condition called *hemochromatosis*. As a result of the action of the **plasmodium malariæ** upon the blood, two very different pigments are formed. There is one which lies within the bodies of the plasmodia, is black in color, gives no iron reaction, and is very little understood, while the other, hemosiderin, is dissolved in the plasma of the blood as a result of the destruction of red blood-corpuscles, and is deposited later in liver, spleen, etc., to produce a *malarial pigmentation* of these organs.

The yellow pigmentation of **jaundice** is a deposition in the tissues of yellow granules or crystals derived from bile in the blood. In many pathological conditions the pigment, which is normally formed in given amount within the cells, may increase—*e. g.*, in the pigmentation of the skin in *pregnancy* and *Addison's disease*, in pigmented *moles*, and the blue-black pigmentation of *melanotic tumors*, etc. Instances of pigmentation due to introduction of *pigment from without* are found in the deep skin pigmentations following the use of large doses of nitrate of silver. *Tattoo markings* and the occurrence of coal-dust in the lungs will serve as further examples.

## TUMORS.

A tumor, or neoplasm, or new growth, is a new formation of tissue, possessing an atypical structure, not exercising any function of service to the body, and presenting no typical limit of growth; or it may be defined as an atypical new formation not the result of "inflammation."

Tumors, as a rule, reproduce with more or less deviation the

structure of the part in which they primarily grow. They are characterized by an independent or lawless growth, their structure is without rule or uniformity, and their history is outside of any physiological limitation. The type of tissue in a tumor may be the same throughout its life history, or it may change to another type within its general class. Tumors are usually sharply circumscribed and well defined from the surrounding tissues, yet, on the other hand, they may shade off so imperceptibly as to make it impossible to place a line of limitation. The new tissues grow imperceptibly, or with such rapidity that their vessels, stroma, and cells are imperfectly formed, thus subjecting such tissues to every retrograde or degenerative change. Tumors usually exist singly, but there may occur simultaneously great numbers of any given type of tumor scattered throughout the body. Different varieties of tumor may coexist entirely independent of one another, or, from one tumor, metastases may form in many distant parts of the body.

All tumors develop by a proliferation or rapid karyomitosis in the cells of the tissue from which they spring, and by a formation of new blood-vessels. Subsequent growth of the tumor is principally by rapid division of the primary tumor cells, yet, less prominently, by the transformation into tumor tissue of the cells of the tissues invaded and of the white blood-cells. Tumors behave differently in their mode of growth. (1) They may grow equally in all their parts and entirely within themselves, so that the surrounding tissues are involved only by pressure. This is called **central growth.** (2) A tumor may grow mainly on the surface, by forcing its way into the intercellular spaces of the tissues about. The tissues thus invaded are also stimulated to proliferation, and a growth takes place both from tumor cell and normal tissue cells. This is called growth by **peripheral infiltration.** (3) Tumor cells may be carried a short way by the blood- or lymph-vessels where they lodge, and develop into tumors which will soon fuse with the mother tumor. This constitutes **discontinuous peripheral growth** in a tumor.

When the tumor cells taken up by the blood- or lymph-vessels are carried to points distant from the primary growth, they lodge and develop into tumors identical in structure with the primary tumor, and we have that very important method of dissemination known as *metastasis.* When we consider the poorly-formed blood-

and lymph-vessels resulting from such rapid development, indeed, the formation of the very vessel wall, at times, by tumor cells, it is not surprising that metastases should form. One cell, detached, will be swept away in the current, lodge in some remote part, and, by rapid cell division, form a secondary or metastatic tumor.

Metastasis through the lymph-vessels usually takes place in those vessels, and in the first set of lymph-nodes which receive lymph from the organ involved; while that through the blood-vessels may occur in any remote organ of the body, but usually is found in the first set of capillaries through which the venous blood must pass in coming from the tumor; hence the frequency, indeed, almost the rule, of secondary tumors of the liver in carcinoma of the stomach.

The retrograde or degenerative changes taking place in tumors are: fatty and myxomatous degeneration, hemorrhage with resulting necrosis or pigmentation, calcification, cyst formation, and cicatrization. Germs may gain entrance and set up a simple or suppurative inflammation in the tumor, or superficial necroses may form ulcers.

Tumors are divided clinically into the **benign** and **malignant**. If by malignancy we mean death-producing, then all tumors may be malignant if located in the right place; as, for instance, a brain tumor or a tumor of the larynx. There are, however, certain characteristics of malignant tumors which divide them sharply from the benign: (*a*) A rapid infiltration of the surrounding tissues without limitation; (*b*) their tendency to ulceration and necrosis; (*c*) a tendency to local recurrence when once removed, dependent undoubtedly upon a failure to remove all tumor cells; (*d*) the formation of metastatic growths in remote parts of the body; and (*e*) a condition of anemia, feebleness, and a diminished general nutrition which is commonly known as **cachexia**. The cachexia seems dependent, in part, upon a diminution in the nutritive supply, by the rapidly-growing tumor, but is, perhaps, most influenced by interfered nutrition and assimilation, due to the location of the growth (esophagus, stomach); or upon loss of sleep, from pain, from degeneration, with suppuration and ulceration. It may, furthermore, be due to various toxic agents produced by the tumor in its growth.

Many **causes** are given for the development of tumors. (1) The most probable explanation is Cohnheim's theory of misplaced or superfluous embryonic cells. According to this theory, certain

embryonic cells have failed to develop, and remain dormant, ready to start up in active growth at any time. When this growth does begin, all the unrestrained activity and force of embryonic tissue is manifested in the midst of adult tissue, where the cells are under the limitation of growth expressed by physiological co-ordination. Nobody has seen such an embryonic cell, but the theory would seem to explain many points concerning tumors, such as their heredity, congenital nature, abnormal and atypical structure, their multiplicity, and their occurrence in tissues of entirely different structure. (2) Trauma certainly seems to be a cause in a certain small percentage of cases, because of the frequency of the carcinomata at points subjected to greatest irritation, as the mouth, pylorus, ileo-cecal valve, and anus. (3) They follow inflammation, especially where there is ulceration or scar formation. Such cases are found in cancer of the stomach, beginning in the edge of a simple ulcer, or in cancer of the gall-bladder, where gall-stones have produced ulceration. (4) There seems to be a balance of forces between the elements of any tissue. With the atrophy of one element, the restraining influence over the other is gone, and the remaining element grows without limitation. Such seems a cause of epithelioma where, in old age, the connective tissue atrophies and the epithelium grows unrestrainedly. (5) A bacterial origin is not proven. (6) Certain protozoa and other cell inclusions are given a place among causative agents, but such connection is not established.

Tumors are best classified according to their embryonic origin. The cells of the embryo are arranged in two layers, the *archiblast* and *parablast*. The archiblast divides (1) into an outer layer, or *epiblast*, which supplies the epithelium of the skin and its adnexa, the epithelium of the terminal portions of the alimentary canal, the nervous system, and the neuroglia; (2) into a middle layer, or *mesoblast*, from which spring the smooth and striped muscle-fibers and the epithelium of the genito-urinary tract; and (3) into an inner layer, or *hypoblast*, which gives origin to the epithelium of the respiratory and alimentary tracts together with all the connected glands. The *parablast* develops later than the *archiblast*, but its origin is not fully understood. From it are formed all connective tissue, including cartilage, bone, teeth, and fat, the blood-vessels and blood-cells, the lymph-vessels and tissues, and the endothelial cells.

## CLASSIFICATION OF TUMORS.

| | | |
|---|---|---|
| | I. *Type of Fully-developed Connective Tissue.* | |
| | *Physiological Type.* | *Tumors.* |
| Parablast | Fibrillar connective tissue. | Fibroma. |
| | Mucous tissue. | Myxoma. |
| | Adipose tissue. | Lipoma. |
| | Cartilage. | Chondroma. |
| | Bone. | Osteoma. |
| | Lymphoid tissue. | Lymphoma. |
| | Neuroglia. | Glioma. |
| | II. *Type of Embryonic Connective Tissue.* | |
| | Sarcoma in all of its varieties. | |
| | III. *Type of Higher Tissues.* | |
| | Muscle. | Myoma. |
| | Nerve. | Neuroma. |
| | Blood-vessels. | Angioma. |
| | Lymphatic vessels. | Lymphangioma. |
| Archiblast | IV. *Type of Epithelial Tissues.* | |
| | Papillæ of skin or mucous membrane. | Papilloma. |
| | Glands, . . . . . | Adenoma. Carcinoma. |
| | V. *Teratomata, or Congenital Mixed Tumors.* | |

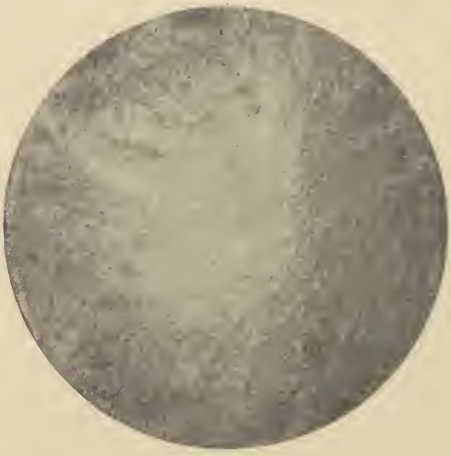

FIG. 14.—FIBROMA DURUM.

The entire structure of the tumor consists of wavy fibers of connective tissue, with very few blood-vessels.

## FIBROMA.

The **fibroma** is a benign tumor composed of fibrillar connective tissue.

*Varieties.*—1. The **fibroma durum** is a hard nodular tumor, tough in consistency, glistening white, and made up almost en-

tirely of thick bundles of fibers in irregular arrangement. Among the fibers are a few flattened or spindle-shape cells and an occa-

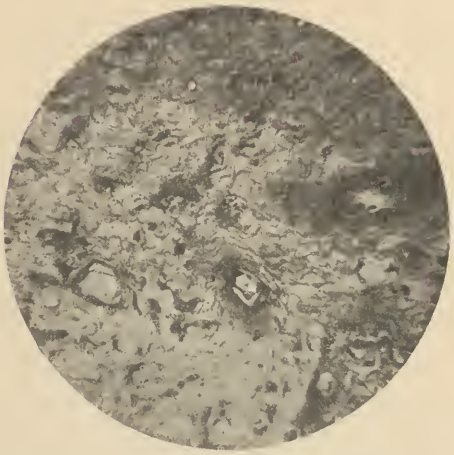

FIG. 15.—FIBROMA MOLLE FROM THE NOSE.
Large, branching cells are numerous; the connective-tissue fibers are loosely arranged, and blood-vessels are numerous.

FIG. 16.—PAPILLOMA OF THE BLADDER.

sional blood-vessel. Examples of tumors of this type are found in the fibromata of the periosteum, uterus, testes, etc.

2. In the **fibroma molle**, round-, spindle,- and branched-cells are abundant. The fibers are few in number, loosely arranged, and

separated by a gelatinous, albuminous material. The tumor is soft, translucent, grayish-white, and contains many blood-vessels. The nasal polypi represent soft fibroma.

FIG. 17.—INTRACANALICULAR FIBROMA OF THE BREAST.
The clear bands represent the lumen of a former duct into which the fibrous projections have grown.

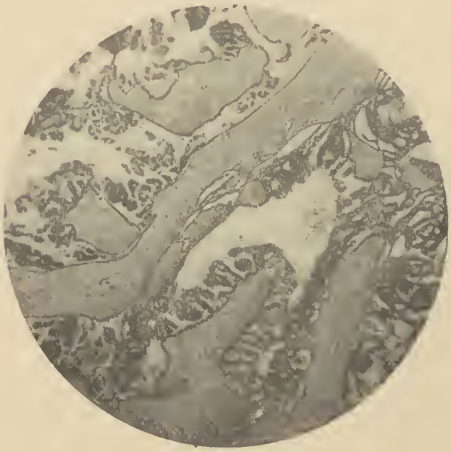

FIG. 18.—PAPILLOMA OF THE OVARY.
Each stalk or branch of the tumor is covered by a single row of cuboidal cells.

3. When a fibroma forms in the skin or mucous membrane, it grows from the papillary layer, and the epithelial cells covering these papillæ keep pace in their growth. This is called a **papil-**

**loma.** The papilloma is often considered as a separate tumor of the epithelial type, and it would seem more accurate to classify it as such in cases of papilloma of the ovary, bladder, intestine, in condylomata, etc., where the connective-tissue element is merely a very thin branching stem and the main element in the tumor is epithelial cells.

4. **Intracanalicular fibromata** are tumors formed within the ducts of glands. Fibrous polypi of irregular shape grow from the walls of the ducts into their lumen. A single layer of epithelial cells covers these polypi, and on section there appears a solid mass

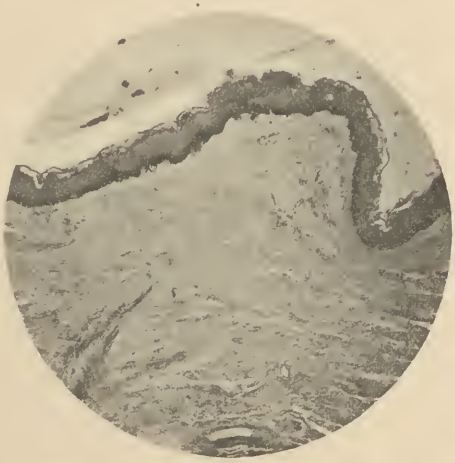

FIG. 19.—KELOID FROM THE SHOULDER OF A NEGRO, WHERE IT OCCURRED WITHOUT PREVIOUS INJURY.

The body of the section is of a firm fibrous structure, and its edge, which represents the surface of the tumor, presents a skin covering containing, in the deeper layer, the black pigment of the negro race.

of connective tissue, divided up by irregular fissures which are lined by a single row of cuboidal cells. These tumors often grow to large size, and occur most frequently in the breast.

5. The **pericanalicular fibroma** is a type of tumor in which the fibrous tissue is deposited cylindrically about the duct but does not grow into the lumen.

6. **Fibroma molluscum** is a name given to multiple, soft, wart-like fibromata occurring in the skin. They are neurofibromata formed on or around the peripheral terminations of the cutaneous nerves.

7. **Keloid** is an irregular, radiating new formation of connective

tissue in the skin and subcutaneous tissue. It has the structure of a hard fibroma and looks like scar tissue, but occurs either spontaneously or in a scar after injury. Keloids are common in the negro.

## MYXOMA.

Connective tissue in its embryonic condition consists almost entirely of spheroidal cells with a small quantity of fluid lying between them. As development advances, these cells become fusi-


FIG. 20.—MYXOMA FROM THE NOSE.

This specimen represents almost a pure myxoma. The blood-vessels (*a*, *a*) consist of a single row of endothelial cells. The dotted portions are homogeneous mucous tissue.

form, branched, and irregular, the fluid increases in amount, and delicate fibrillæ run through it. To this older form of embryonic connective tissue is given the name of *mucous tissue*. It is found normally in the vitreous of the eye and in the umbilical cord of the fetus. The **myxoma** is a benign tumor, composed of mucous tissue with irregular, fusiform, branching, and anastomosing cells, and a gelatinous intercellular substance which is coursed by fine fibrillæ. They are soft, translucent, and usually very vascular. A white precipitate is formed when dilute acetic acid is applied to the cut surface. Few tumors are myxomatous throughout, but this tissue is often found in combination with others, forming thus fibromyxoma, lipomyxoma, chondromyxoma, and myxosarcoma.

*Seat of Occurrence.*—Myxomata develop in fat tissue, subcutaneous and submucous tissue, in the marrow and periosteum of bone, in the breast, and in the parotid glands.

Myxomata are essentially benign, but many myxomata of the nerves are certainly malignant, and other varieties are often considered as relatively malignant tumors because of the frequency with which they change into sarcomata.

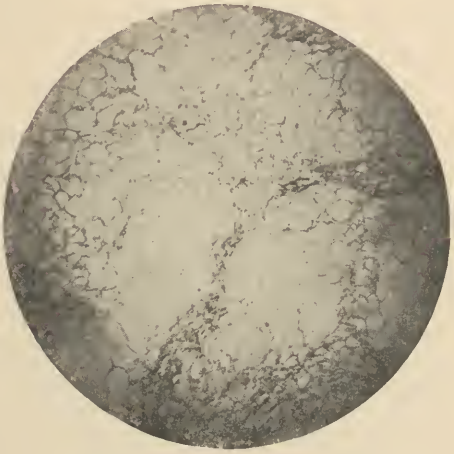

FIG. 21.—LIPOMA OF THE BUTTOCK.

## LIPOMA.

A lipoma is a benign tumor composed of fat tissue. The structure of such a tumor varies in no way from normal fat tissue, except that the fat cells are usually larger and less regularly arranged. They are usually encapsulated. Associated with other tissues they form lipofibroma, lipomyxoma, angiolipoma, etc.

*Seat of Occurrence.*—They occur in the subcutaneous tissue of the neck, buttocks, back, axilla, abdomen, thigh, and occasionally in the abdominal cavity, breast, and kidney.

## CHONDROMA.

The chondroma is a hard, spherical, or knobbed tumor, made up of hyaline, elastic, or fibro-cartilage, but more frequently of a combination of the three. The number, size, form, and arrangement of

the cells varies greatly in the different tumors,—sometimes, indeed, in the same tumor,—but the uniformity in structure of normal cartilage is lost. The *cells* may be numerous or few, small or large, or both, and frequently are fusiform or branched. They have a capsule or not, and sometimes lie in groups in a mother capsule. With other tissues they form osteochondroma, myxochondroma, and chondrosarcoma.

*Seat of Occurrence.*—They occur most frequently in places where cartilage normally exists, and are called *ecchondromata.* When occurring in tissues other than cartilage—as, for instance, in the

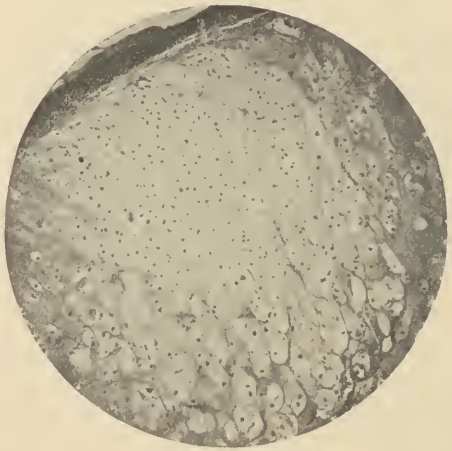

FIG. 22.—CHONDROMA.

The dark band above represents the capsule of the tumor. Cartilage cells of every type are seen in the capsules, and fibrous tissue supports these capsules.

periosteum, testicle, parotid, and mamma—they are called *enchondromata.*

Clinically, they are benign, as a rule, but metastases sometimes form in the lung and heart.

## OSTEOMA.

An osteoma is a tumor made up of bone. It may be soft and spongy, like cancellous tissue, or denser (eburneous), resembling compact bone, or very hard and dense, like ivory (*ivory exostoses*). If the new growth is diffusely spread out it is called a **hyperos-**

**tosis**; if confined to a limited area, an **osteophyte**, or, if of considerable size, an **exostosis**. Circumscribed bony growths inside of bones are called **enostoses**. Of the types of bony tumor not connected with old bone are: (1) Those surrounded by the periosteum, yet separate from the bone; (2) those located near a bone; (3) those located in muscles and tendons remote from bones; (4) those occurring in the lungs, meninges, diaphragm, skin, and parotid gland.

## ODONTOMA.

Tumors containing dentine are sometimes formed from the pulp, during the development of the teeth. These are called **odontomata**.

## ANGIOMA.

The true angioma includes those new growths in the structure of which blood- or lymph-vessels constitute such an important part as to determine the character of the tumor. Hence we have: (1) **Hemangioma**, or a blood-vessel tumor; (2) **lymphangioma**, or a lymph-vessel tumor.

**Hemangiomata** are of four types: 1. The **simple angioma**, or **angioma telangiectoides**, is a circumscribed dilatation of pre-existing or newly-formed capillaries, with thin or thick walls. These walls are imbedded in a more or less abundant connective-tissue stroma. The dilatations are cylindrical, fusiform, and sacculated; there is abundant anastomosis and a perfect tangle of intertwined vessels. These tumors are popularly known as *strawberry marks*, or *vascular nevi*.

2. The **simple hypertophic angioma** is a form of tumor composed of dilated *capillaries*, but the capillary wall is disproportionately thick, and is rich in cells, like the wall of an arteriole.

3. In the **cavernous angioma** the structure is that of a system of wide, variously-shaped, intercommunicating cavities, separated from one another by mere connective-tissue partitions. They are dilatations of pre-existing vessels, and are lined with endothelium. The structure is that of the corpora cavernosa of the penis. They occur on the skin as raised patches of bluish-red color, in the liver, and, less frequently, in the brain, bone, spleen, kidney, uterus, etc.

4. The **cirsoid aneurysm** is a condition where a whole system of arteries are dilated, tortuous, and thickened, forming one large

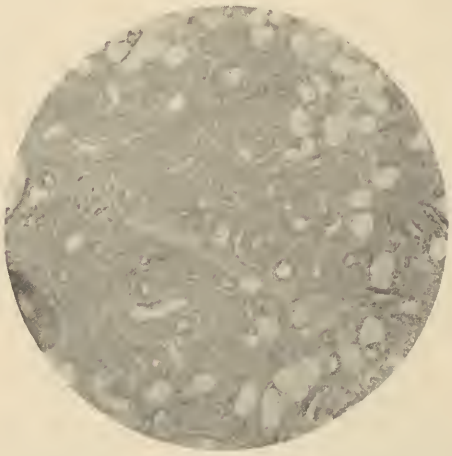

FIG. 23.—ANGIOMA TELANGIECTOIDES FROM THE BACK OF A CHILD.
The section shows a tangle of thickened blood-vessels imbedded in fat, which is seen as the clear spaces around the border.

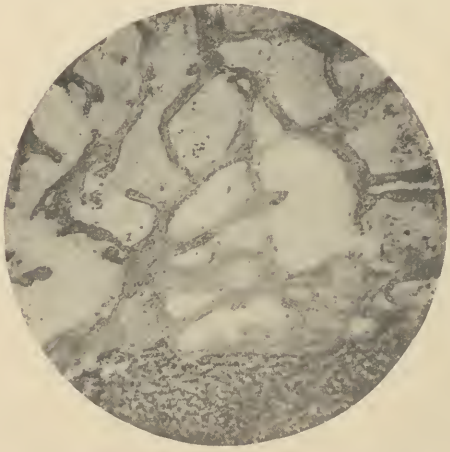

FIG. 24.—ANGIOMA CAVERNOSUM OF THE LIVER.
Liver tissue is shown below, and the large sinuses above marked off by fibrous septa represent the typical structure of such a tumor.

mass. The tumor feels like a bunch of earthworms beneath the skin.

**Lymphangiomata** are composed of old and new lymph-chan-

nels, dilated to form either simple dilatations or cystic or cavernous spaces. They are identical in structure with hemangioma, except that one is filled with lymph and the other with blood; furthermore, the walls of the lymphangioma are thinner and more delicate.

*Seat of Occurrence.*—They occur in the skin of the scrotum, vulva, and neck. In the thigh they form the condition of elephantiasis; in the tongue, that of macroglossia.

Under the head of lymphangioma must be included certain other new formations occurring in the skin. They are: (*a*) The **pigmented nevi**, which appear as pale or dark-brown plaques on a level with the surface of the skin or as elevated warty growths; (*b*) **lentigines**, or permanent pinhead spots of brown or yellow pigment; (*c*) **freckles**; and (*d*) the **fleshy warts**, which are well-defined non-pigmented elevations springing from the papillæ and having smooth or uneven contour. They are covered by normal or hypertrophic epithelium.

The structure common to all of these conditions is that of a connective-tissue framework inclosing masses of cells in groups or bands. These cells lie partly in the papillæ, partly in the corium of the skin, and in the pigmented forms they contain pigment granules. The cells seem to be pathologically developed from the endothelium of the lymph-vessels; hence their classification among the lymphangiomata.

## MYOMA.

Myomata are tumors composed of newly-developed muscle-fibers. They are divided into **leiomyomata**, formed of smooth muscle-fibers, and **rhabdomyomata**, formed of striped muscle-fibers.

**Leiomyomata** are characterized by fusiform smooth muscle cells, which are of uniform size, have elongated or rod-shaped nuclei, and are regularly arranged in bands or parallel lines. These cells are packed closely together, interlace in every direction, and are intermingled with a certain amount of connective tissue and a varying number of blood-vessels. When the connective tissue is in large amount, it is termed a fibromyoma.

*Seat of Occurrence.*—They occur as nodular tumors in the uterus, intestinal tract, bladder, and prostate.

The **rhabdomyomata** are very rare tumors, made up of striated

muscle tissue. The muscle-fibers of such a tumor are nucleated and ill-developed, slender, spindle, club-shaped, or round in form, and very irregularly arranged. They are variously striated in a longitudinal or transverse direction and are intermingled with connective tissue, which varies in amount in different tumors. The sarcolemma is absent.

*Seat of Occurrence.*—They occur in the kidney, testicle, and ovary as circumscribed nodules. Clinically, they are benign.

FIG. 25.—GLIOMA FROM BRAIN. (*Teased preparation.*)
The diagram shows the delicate branches of the cells and the fibrous intercellular substance.

## GLIOMA.

Gliomata are tumors which grow from, and consist essentially of, the glia- (connective-tissue) cells of the central nervous system, the ganglion-cells taking no part in the tumor formation. The structure is of an extremely delicate network of fibers, among which are imbedded numerous small round-cells with disproportionately large nuclei. The cell protoplasm is distinguished with difficulty, but teased preparations show great numbers of fine branching processes from the cells, which make up the delicate fibrillar network. The blood-vessels are highly developed. These tumors are soft and can not be sharply defined from the surrounding brain tissue in color or consistency. They are very frequently associated with other tumor tissues, as gliosarcoma, gliomyxoma, etc.

*Seat of Occurrence.*—They occur in the *retina*, brain, and spinal

cord. The glioma occurring alone is benign, but that form found in the retina is very malignant and results fatally unless enucleation is early. This leads to its classification as a sarcoma; indeed, it is claimed by some that all gliomata are neurogliar sarcomata.

**Neuroglioma ganglionare** is a name applied to another tumor of the central nervous system. It is an ill-defined or circumscribed tumor made up of glia-cells, ganglion-cells, and nerve-fibers. The structure is usually that of a more or less dense glia tissue, with a few nerve-fibers and ganglion-cells scattered throughout it. It has the same clinical features as glioma.

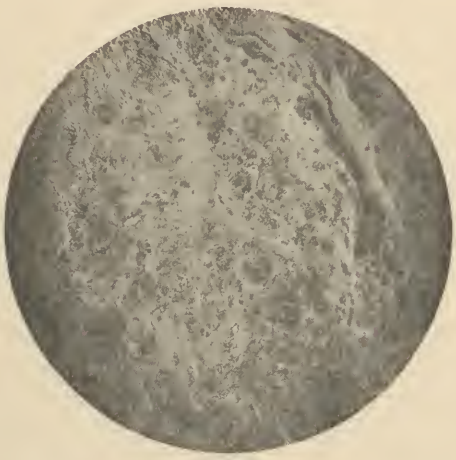

FIG. 26.—AMPUTATION NEUROMA FROM THE THIGH.

## NEUROMA.

Most tumors described as neuromata are nothing more than fibromata of the nerves. Such, for example, are the tumors in the ends of amputated nerves, tumors in the course of nerves, and multiple tumors present in different parts of the body. In such neurofibromata there is a growth in the connective tissue of the outer, sometimes of the inner, layers of the endoneurium, so that the nerve-fibers and bundles lie inclosed in a connective-tissue mass. True neuromata are either ganglionic or fibrillar. The **ganglionic type** of neuromata is only occasionally found in certain teratomata of the ovary, testicle, and sacral region. The **cirsoid** or **plexiform neuroma** represents the fibrillar neuroma, and is made up of medullated nerve-fibers. The fibers reveal extensive fibromatosis,

from excessive growth of the endoneurium; but, aside from this, the nerve-fibers are not only greatly thickened and increased in number, but are increased in length and rendered tortuous, until we have a mass of curled and intertwined nerve bundles.

*Seat of Occurrence.*—They occur in the head, body, and extremities. Both neurofibromata and true neuromata sometimes take on a sarcomatous character. Hereditary transmission and congenital predisposition have been established in both forms.

## LYMPHADENOMA.

Lymphadenoma (or *lymphoma*) is a term applied to tumors arising from a proliferation of the lymphadenoid tissue already existing in certain organs of the body. Such proliferation may affect whole groups of lymph-nodes, the tonsil, and the spleen, as in the general diseases of leukemia and Hodgkin's disease, or it may be limited to the tonsils, even to the pharyngeal tonsil, as in *adenoids* of children. Microscopically, there is an enormous increase in the number of uni- and polynuclear cells and in the reticular tissue. The character of the lymphatic tissue is preserved, but the normal relations of follicles, cords, and lymph-sinuses are lost. The cause of such development is unknown, but the fact that they are not hypertrophic classes them as tumors.

*Seat of Occurrence.*—The types are leukemia, pseudoleukemia (Hodgkin's disease), and tonsillar tumors.

## SARCOMA.

A sarcoma is a connective-tissue tumor in which the cellular elements are much more prominent, in both number and size, than the intercellular substance. This characteristic stamps them as undeveloped or embryonic connective-tissue tumors. All epithelial structure is completely absent or only accidentally present. The intercellular substance, however, is always present and in intimate relationship with the cells by direct fibrillar processes. This will be well illustrated by stating that should an attempt be made to pencil out the cells in water, it would be found impossible, because each cell would hang to the intercellular connective tissue

by a fine fibrillar process. The cells are most varied in shape and size, but the one type of cell usually predominates in a given tumor to furnish a suitable qualifying name.

The blood-vessels form a constant and important element in the structure of sarcomata, and are intimately associated with both cells and intercellular substance. In fact the tumor cells oftentimes form the very walls of the blood-vessels, and from this intimate relationship it can easily be understood why metastases should form almost entirely through the transportation of sarcomatous cells by the blood-current. Cysts frequently form. The overlying skin is not involved.

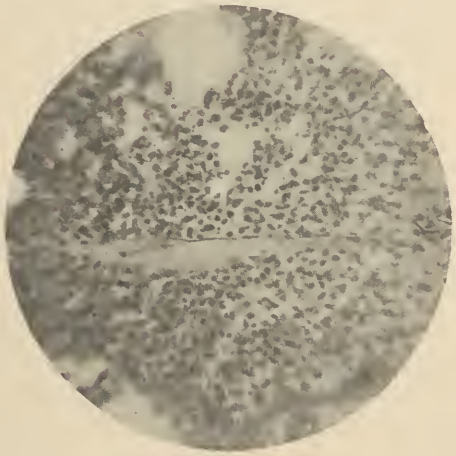

FIG. 27.—SMALL ROUND-CELL SARCOMA.
The clear space below the center of the diagram is a poorly-formed blood-vessel.

*Clinically*, their cellular character, vascularity, rapid growth, marked tendency to local recurrence, the formation of metastases, and the production of the cachectic state, all go to stamp the sarcoma as the most malignant of tumors. They *occur in youth* and early middle life, affecting organs and tissues which are developing or in active function, while carcinoma belongs to later life and senescent tissues.

The **small round-cell sarcoma** is made up of very small round-cells which have very little protoplasm, and a single, relatively large oval or round nucleus. The intercellular substance is usually scanty and delicately fibrillated or granular. The vessels are numerous, the tumors soft, juicy, and very malignant.

Such tumors occurring in lymph-nodes are called lymphosarcomata, and these differ in no way from lymphomata except that they invade the surrounding tissues and form metastases.

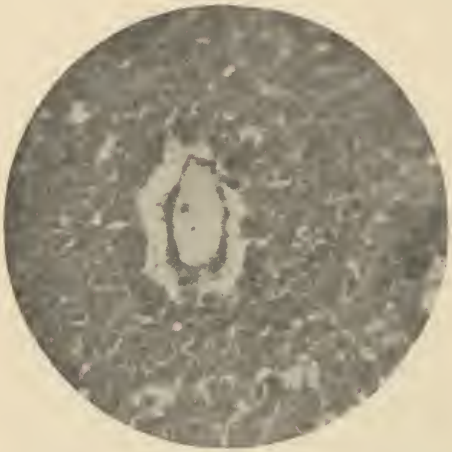

FIG. 28.—LARGE ROUND-CELL SARCOMA.
The clear space in the center is a blood-vessel with detached endothelium.

*Seat of Occurrence.*—They occur in the periosteum, bone, lymph-glands, subcutaneous tissue, testicle, eye, ovary, etc.

In the **large round-cell sarcomata** the cells are very large, abundant in protoplasm, and have from one to three very large round or

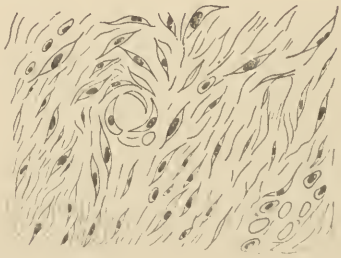

FIG. 29.—SMALL SPINDLE-CELL SARCOMA.

oval nuclei. They usually have polymorphous cells as well. The intercellular substance is present in varying amount, the vessels are abundant, the tumor is firmer and less malignant than the preceding form.

*Seat of Occurrence.*—They occur in much the same regions as the preceding, but are found especially in the subcutaneous tissue of the pharynx and posterior nares, forming small, firm, pale polypi.

The **small spindle-cell sarcoma** is a firm, dense, and elastic tumor, less malignant and of more frequent occurrence than any other form. The intercellular element may be small in amount, or so abundant as to constitute a fibrosarcoma. The cells have a varying amount of protoplasm, are regularly formed, contain an oval nucleus, and are arranged either in bundles parallel to one another or they interlace intricately.

*Seat of Occurrence.*—They are found in the periosteum, subcutaneous tissue, uterus, mamma, testicle, thyroid, etc.

In the **large spindle-cell type** the cells are relatively thick, irregular, or bifurcated, the tumor is softer, pinker, and more vas-

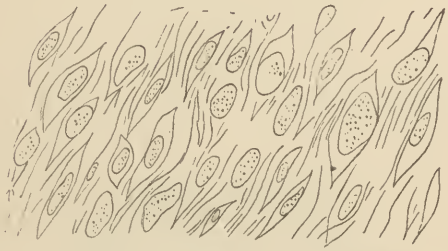

FIG. 30.—LARGE SPINDLE-CELL SARCOMA.

cular, the growth is more rapid, and metastases form early in the lymph-glands.

*Seat of Occurrence.*—They occur most frequently in the skin.

**Melanosarcomata** are usually of the spindle-cell type, but the characteristic point is the deposition around the nuclei of the cells, less frequently in the intercellular substance, of granules of brown or black melanin pigment. The pigment is distributed through the tumor in streaks or patches and varies in amount.

*Seat of Occurrence.*—Such pigmented tumors arise in connection with the choroid of the eye or moles of the skin. They are the *most malignant of tumors* and form rapid metastases.

**Myeloid** or **giant-cell sarcomata** occur most frequently in connection with bone. They arise in the marrow and periosteum, form spheroidal or fusiform tumors of the small spindle variety,

and are characterized by the presence of numbers of large multinuclear or giant-cells. These giant-cells appear as irregular areas of delicate protoplasm containing from 10 to 20 or more nuclei. When

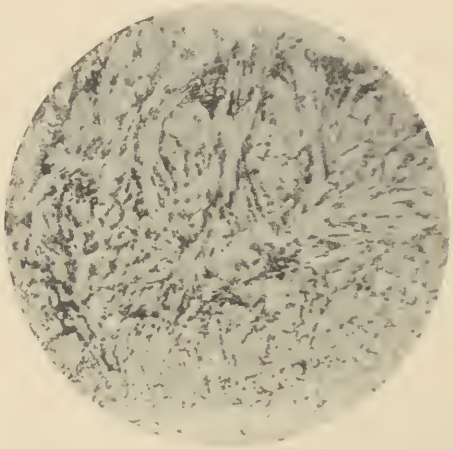

FIG. 31.—MELANOSARCOMA OCCURRING IN A SPINDLE-CELL TUMOR.
The melanin pigment is deposited in streaks.

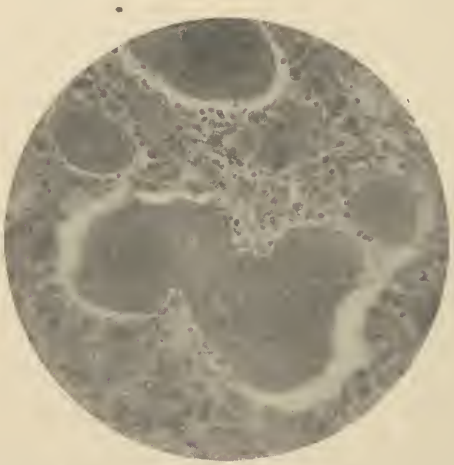

FIG. 32.—MYELOID OR GIANT-CELL SARCOMA OF THE TIBIA.
Large round-cells are associated with the giant-cells.

they arise from the marrow they are apt to be very soft and vascular and cause resorption of the bone. The periosteal form is much denser. They grow very slowly and are the least malignant of sarcomata.

*Seat of Occurrence.*—They occur most frequently in the lower jaw, lower end of the femur, and head of the tibia.

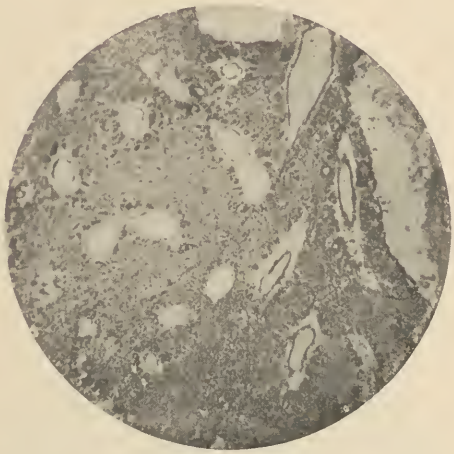

FIG. 33.—ANGIOSARCOMA.
The vessel wall consists merely of endothelial cells, which are detached from the surrounding large round-cells of the tumor.

FIG. 34.—ALVEOLAR SARCOMA FROM THE SUPERIOR MAXILLA.
The arrangement in alveoli seems dependent upon a growth of cells from the walls of lymph-vessels.

In the **angiosarcomata** the tumor is almost wholly made up of a tangle of vessels whose walls are surrounded by heavy masses of proliferating round-cells, which extend even to the endothelium.

The vessels anastomose freely, are packed closely together, and may be fused in such a way that the tubular structure is wholly lost. They are called *perithelial sarcomata.*

*Seat of Occurrence.*—They occur in the brain, testicle, lymphatic glands, breast, skin, and bone.

**Alveolar Sarcoma.**—In certain sarcomata the intercellular substance is more or less abundant and arranged in a wide-meshed net with masses of cells in the meshes. This gives to us the alveolar structure which is so characteristic of carcinoma. The cells, however, do not lie loose in the meshes but are intimately related to the intercellular substance which sends fine trabeculæ into the

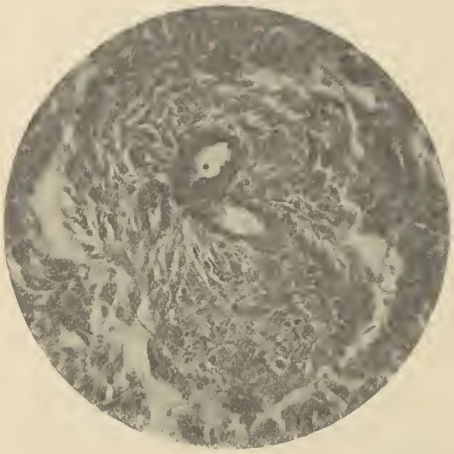

FIG. 35.—ENDOTHELIOMA OF THE DURA MATER.
Two blood-vessels are seen in the center and from the endothelial cells of these the surrounding mass of cells have proliferated.

alveoli between the cells. In water, the cells of a carcinoma could be pencilled out of the alveolus, while in alveolar sarcoma the fibrillæ would hold each cell in place. The new formation of blood-vessels and their arrangement are also diagnostic. Clinically, they are very malignant tumors.

*Seat of Occurrence.*—They occur in the skin, lymph-nodes, bone, and the pia mater.

The **endothelioma** is a tumor arising from a proliferation of the endothelial cells lining the lymph-vessels and lymph-spaces. The cells making up such a tumor are either of almost normal character, very large and thick, or even cylindrical and cuboidal. Sometimes

the cells are packed together in dense and concentric masses of glistening appearance and contain cholesterin crystals, when they are called **cholesteatoma.** Arising from the lymph-vessels as they do, it can readily be seen that a tubular structure is often given to the tumor resembling carcinoma. The periphery of the tumor, however, will show a proliferation from the endothelium, which is diagnostic.

*Seat of Occurrence.*—They occur in the pia and dura mater, pleura, periosteum, and mamma.

**Cylindroma.**—This name is given to those sarcomata in which layers of flattened or cuboidal cells are arranged about a central homogeneous or striated cylinder of hyaline degeneration.

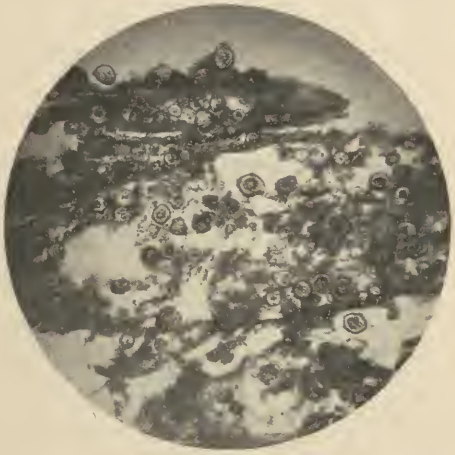

FIG. 36.—PSAMMOMA.
The circular markings represent the calcareous deposits.

The **myxosarcoma** is one in which myxomatous change has taken place, but this must be considered a degeneration in a tumor rather than a separate type. The same is to be said of **osteosarcoma,** where bony trabeculæ without regular lamellation and without Haversian canals form in the substance of a periosteal spindle-cell sarcoma.

The **chloroma** is a lymphosarcoma arising in connection with the skull and presenting a bright green color on fresh section. The color is not understood.

In certain sarcomata of the pineal body and choroid plexus, small, sand-like areas of calcification are often formed. Such tumors are called **psammomata,** or brain sand.

## EPITHELIAL TUMORS.

In the tumors described thus far there has been a connective-tissue origin or a development from the parablastic germ layer. We have now to consider certain new growths springing from some one of the archiblastic layers and having epithelial cells as their characteristic and predominant element. Such epithelial tumors have certain general characteristics in common. Besides the cellular element which characterizes these tumors, there is also a connective-tissue stroma which supports the cells and carries the vessels. The connective tissue is abundant or slight, and is usually arranged to form the walls of variously-shaped alveoli within which the epithelial cells lie. These cells, however, have no connection with the stroma, and are separated from one another by cement substance alone. Some of the tumors are benign (the adenomata), others are very malignant (the carcinomata and epitheliomata), while on the border line between these two a distinction oftentimes can not be made.

### ADENOMA.

In the adenoma the reproduction of typical gland tissue is maintained. The acini are regularly formed and usually have a lumen. The cells are arranged in a single row upon a basement membrane of endothelial cells with a double contour, but are not of uniform shape, nor do they conform to the shape of the cells of the gland from which they originate. *Simple hypertrophy* of a gland, with increased connective tissue, *does not* constitute an adenoma. There must be a *new* formation of more or less typical gland tissue. Furthermore, there must be a lack of conformity to the gland tissue from which it originates, both in anatomical character and mode of growth. There must be a proliferation of epithelium, which is followed by the formation of gland sprouts, and these sprouts, penetrating into the surrounding tissue, cause a connective-tissue proliferation as well. These tumors are usually circumscribed, nodular, and of tough consistency, except in the papillary forms.

*Seat of Occurrence.*—They occur in the mamma, ovary, liver, kidney, thyroid, prostate gland, and in the mucous membrane of the gastro-intestinal tract and uterus.

In the **adenoma papilliferum** there is a rapid growth of con-

nective-tissue papillæ from the walls of the gland tubules beneath the epithelium. A correspondingly rapid growth of the epithelium in a single layer takes place to cover these outgrowths.

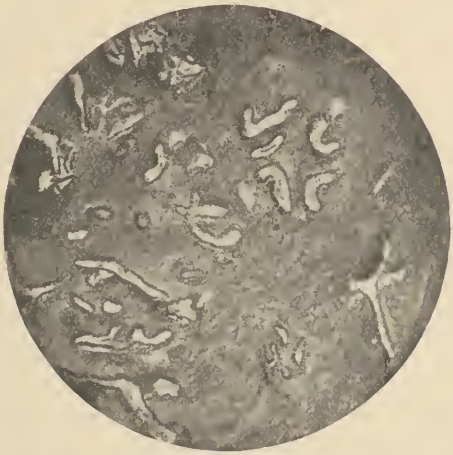

FIG. 37.—FIBRO-ADENOMA OF THE BREAST.
The clear areas represent gland acini, lined by a single row of cells.

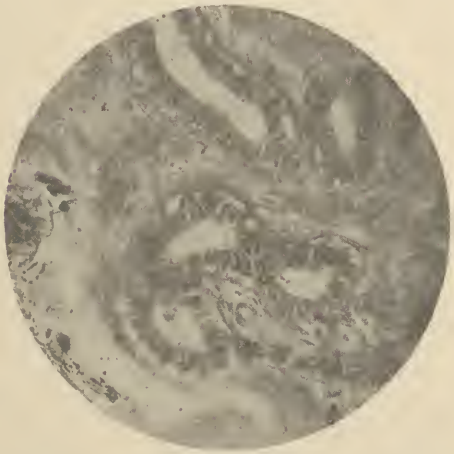

FIG. 38.—ADENOMA OF THE BREAST. (High power.)
In this tumor the gland tissue predominates and the stroma is loose and cellular.

The **multilocular cystoma**, or glandular cystadenoma, is one of the most important forms of adenoma. They form very large tumors, made up of innumerable small and large cysts with

smooth glistening walls, and lined by a single row of columnar, and often ciliated epithelium. The fluid contained is clear or clouded, thin or thick, and tenacious. These tumors have their origin in an ingrowth of epithelium, covering the ovary into the stroma of the organ, and are characterized by a marked tendency to form cysts. Coincident with the formation of the cyst, or at a later date, minute or large cauliflower papillary excrescences may be formed within some or all of the cysts. They are then called **papillary cystoma**, and are regarded by some authors as a separate type of cystadenoma.

*Seat of Occurrence.*—These tumors occur in the ovary, breast, testicle, kidney, or liver, and may take on a malignant form.

Adenomata are differentiated with difficulty from many *glandular hypertrophies* occurring in the mucous membrane of the intestine and uterus. These seem to be atypical gland structures, developed as a result of great reproductive activity.

The adenomata are benign tumors in general, but some of the adenomata of the stomach and intestines must be classed among the most malignant of tumors. Their rapidity of local extension, invading all adjacent tissues, the formation of metastases, and the production of cachexia would place them in this class. These have been called **adenoma destruens** or **malignum.** Their structure is that of very irregular tubular glands, with high columnar cells arranged in a single row on a basement membrane and supported by a very slight connective-tissue stroma. Furthermore, certain adenomata begin as such, but later take on a true cancerous nature. These are called *adenocarcinoma.* Thoma believes that a great number of carcinomata of the breast come from simple adenomata.

## CARCINOMA.

Carcinomata are characterized by a perfectly atypical arrangement of the proliferating epithelial cells in variously-shaped alveoli, clusters, and columns, and by their tendency to unlimited growth. The growth of carcinomatous cells undoubtedly proceeds from a rapid mitotic cell-division, taking place first in the old gland cells and later in the newly-developed tumor cells. The arrangement of the cells in alveoli varies greatly. There may be huge masses of cells surrounded by small bands of connective-tissue stroma,

alveoli containing small numbers of cells in no regular arrangement, long columns of cells extending into the tissues, or cells in groups of two or three wedged between dense bands of fibrous tissue. Never is there connective tissue between the cells *within* the alveoli. There is **no typical** and **diagnostic cancer-cell.** On the other hand, there is great polymorphism of cells, undoubtedly dependent upon their origin, rapidity of growth, and certain conditions of nutriment and pressure. Thus, in cancer of the intestine we may have cylindrical cells as the characteristic form in the tumor; of the skin, stratified epithelium; of the breast, cuboidal cells; while in other parts of any one of these tumors may be found small round-cells, young cells, or cells distorted and flattened by pressure. It is the general character of the cells, together with the topography, seat, and clinical history of the tumor, upon which we base a diagnosis.

These tumors possess all the characteristics of malignancy. Their peripheral extension seems to be chiefly through the lymph-spaces, but the septa and sheaths of nerves provide a very certain means of dissemination. *Metastasis* occurs through the lymph-channels as distinguished from sarcoma, where it takes place through the blood-current; and, consequently, the first lymph-nodes in the chain receiving lymph from the part involved will be earliest infected. Infection may be carried, however, by the blood-vessels, and from nodular tumors of identical structure in distant organs, as is evidenced by the regular appearance of secondary tumors of the liver in cases of cancer of the stomach where the portal vein is the carrier.

Retrograde changes come on early. They consist in fatty degeneration, calcification, and, most frequently, necrosis with gangrene and ulceration.

The *cause* of cancer has not been arrived at as yet. By histological investigation we find that the growth of cancer is due to a pathological penetration of epithelium into connective tissue. This may be due to diminished resistance of the connective tissue or to an increased proliferating power of the epithelium, but most probably it is due to both. As yet, no evidence justifies the assumption that parasites are the cause of the proliferation. That chronic irritation plays a part is shown by its occurrence at the orifices of organs and of the body, and in pipe-smokers, tar and paraffin workers.

**Epitheliomata** arise from the cells of the stratified or squamous

epithelium of the skin and mucous membrane. The cell growth, instead of being upward from the limiting membrane toward the surface, has been reversed. The interpapillary cells have increased

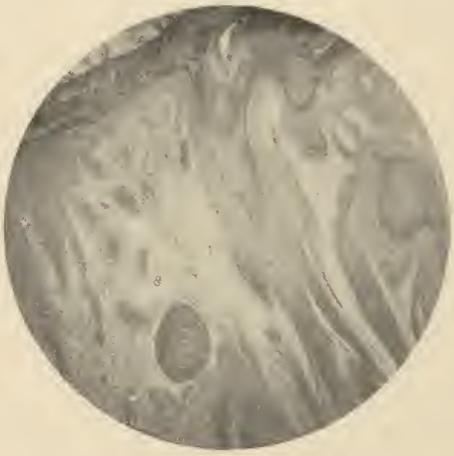

FIG. 39.—EPITHELIOMA OF THE LIP.
The skin surface is above and columns of epithelial cells descend deep into the tissues. Two typical "pearls" are seen.

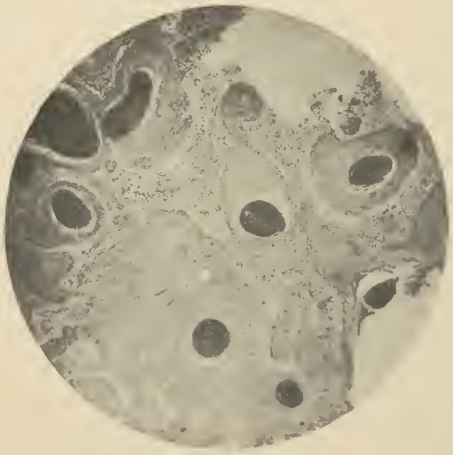

FIG. 40.—EPITHELIOMA OF THE CLITORIS.
Many pearls are seen imbedded in separate masses of cells.

in number, then broken through into the subcutaneous layers, and epithelial cells in columns and in nests are found in the tissues beneath the skin. The cells are of every variety found in the

normal skin, characteristic among which are the "prickle cells." The life history of the cell being the same and the growth being downward instead of upward, the thin, dry, horny epidermis cell is

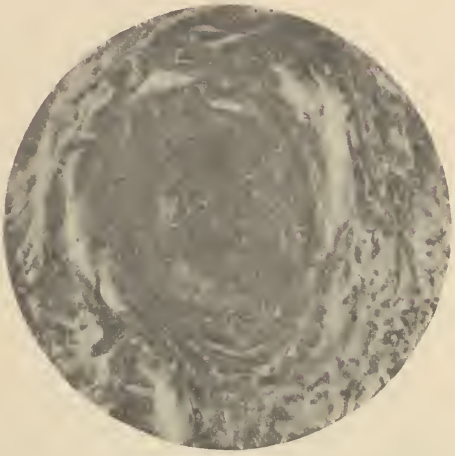

FIG. 41.—AN EPITHELIAL PEARL OR CELL NEST.
Showing the lamination and cellular character.

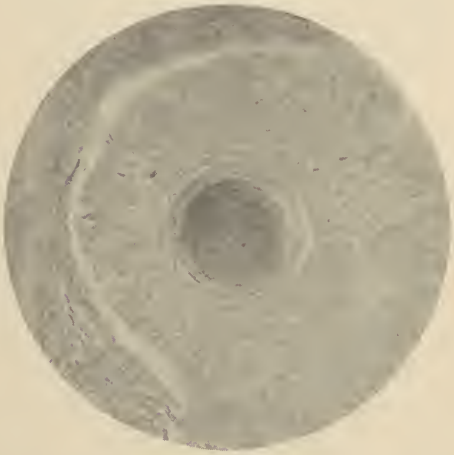

FIG. 42.—EPITHELIAL PEARL WITHIN A MASS OF CANCER-CELLS FROM A FUNGOUS EPITHELIOMA OF THE CERVIX.
The fibrous strip below represents the slight stroma of the tumor.

found deep in the tissues, and they are often packed together concentrically, like the layers of an onion, to form the "*epithelial pearls*" or "cell nests." The outermost cells of such a pearl have nuclei,

but these disappear as the center is approached, where we find only the flat, horny cells. Such pearls or nests are a characteristic feature. Giant-cells may be present. Metastatic infection of the lymph-nodes is early, except in the superficial form.

*Seat of Occurrence.* — Epitheliomata occur most frequently at points of junction of the mucous membrane and skin,—as the lip, nose, labium, glans penis,—and are frequent in the mouth, esophagus, vagina, and cervix uteri.

Certain **superficial forms** of epitheliomata (*rodent ulcers*) occurring on the face have some very different characteristics. Small, cuboidal cells seem to grow down in anastomosing columns

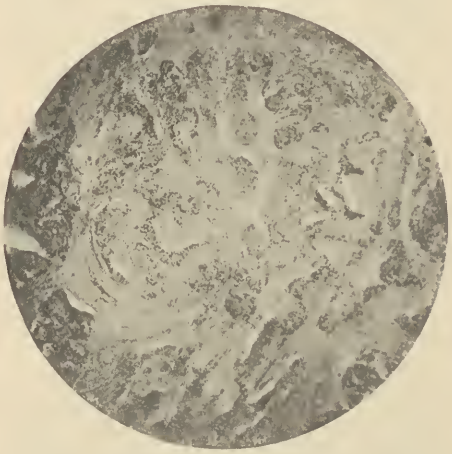

FIG. 43.—CARCINOMA SIMPLEX OF THE BREAST.
The cells and stroma are about equal in amount, and the cells are arranged in alveoli in small masses.

from the deep layers of the rete, to form what are sometimes called *tubular* epitheliomata. Their origin is claimed by some to be in the sweat glands. The cell masses grow very slowly (for years), and connective tissue may form the predominant element. They do not infect the lymph-nodes and are not very malignant.

*Seat of Occurrence.*—They occur on the nose and other parts of the face.

**Carcinoma simplex**, or gland-cell carcinoma, usually occurs in glands, and forms hard, nodular tumors. The cells have no constant characteristic shape, and are arranged in alveoli of different

shapes and sizes, which are formed by a strong connective-tissue stroma. The cut surface is of a grayish-white color.

**A scirrhous carcinoma** is one in which the cellular element is

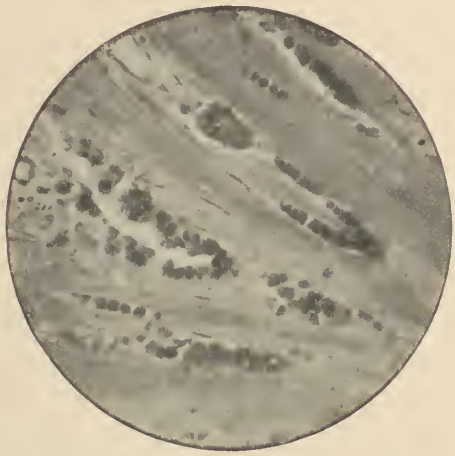

FIG. 44.—SCIRRHOUS CARCINOMA OF THE BREAST.
The cancer-cells are arranged in collections, in rows, or are found singly in the dense fibrous tissue.

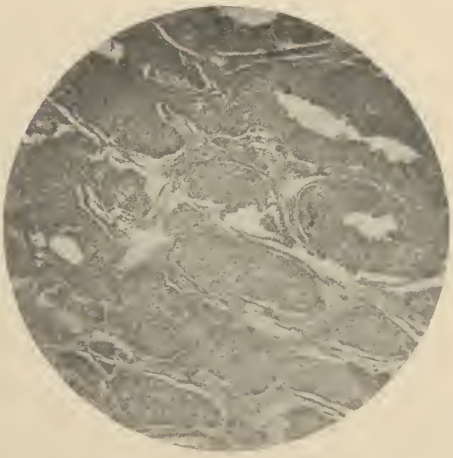

FIG. 45.—MEDULLARY CARCINOMA OF THE BREAST.
The stroma is represented by mere lines between the masses of cells.

relatively small and the main mass of the tumor is made up of tough fibrous tissue. There is no strict line of division between this and carcinoma simplex, the difference being mainly one of degree.

*Seat of Occurrence.*—They occur in the breast, stomach, and sometimes in the testicle and ovary.

In the **medullary carcinoma** there are very large numbers of

FIG. 46.—COLUMNAR-CELL CARCINOMA OF THE STOMACH.
The newly-formed ducts are lined by high columnar cells.

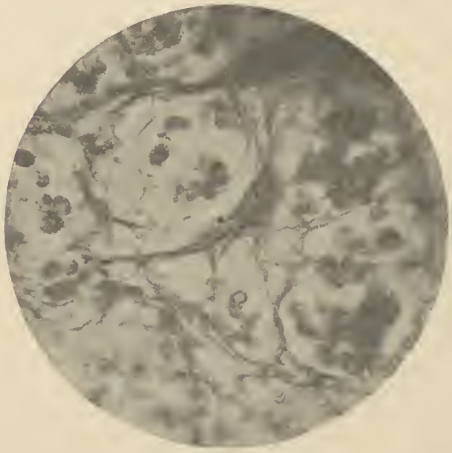

FIG. 47.—COLLOID CARCINOMA.
The clear portions represent the areas of colloid degeneration, and only a few cancer-cells are left in each acinus.

cells, arranged in alveoli without a lumen. The stroma is delicate, and the consistency of the tumor soft and juicy.

The **cylindrical-cell carcinomata** occur in the mucous mem

brane of the intestine and uterus; also in the gall-ducts and respiratory passages. They form soft nodular or papillary tumors, which are rich in cells. The arrangement is of high cylindrical cells lining the regular alveoli, while the remainder of the cells are variously arranged. The comparatively complete development of the epithelial cells, and their arrangement in alveoli, often render it difficult to differentiate this tumor from benign adenoma, but, as a rule, the cells have in some part broken through the basement membrane and appear in irregular masses, infiltrating the deeper coats.

**Colloid carcinoma** is a form in which the cancer-cells have undergone a colloid degeneration. This colloid material is deposited in droplets or masses, giving the entire tumor a transparent appearance. When the degenerative process is extensive, the stroma about the alveoli shows very distinctly, and the term *alveolar carcinoma* is applied.

*Seat of Occurrence.*—Such tumors occur in the intestine and breast, sometimes in the ovary.

In the **carcinoma myxomatodes** the stroma of the tumor is made up of mucous tissue. It occurs in the intestine and mamma.

DIFFERENTIAL DIAGNOSIS OF SARCOMA AND CARCINOMA.

| | Sarcoma. | Carcinoma. |
|---|---|---|
| Occur. | In youth and before forty. | After forty years. |
| Origin. | Parablastic layer. | Archiblastic layer. |
| Stroma. | Between each cell and connected with cells. Does not form alveoli. | Forms alveoli surrounding masses of cells. |
| Cells. | Embryonic or granulation tissue. No epithelial cells. | Epithelial cells of various shape and size. |
| Intercellular Substance. | Stroma. | Cement substance alone. Never stroma. |
| Vessels. | Embryonic, and in definite relation to cells which make up walls. | Well developed. In walls of alveoli and never in contact with cells. |
| Metastasis. | By blood-vessels. | By lymphatics. |

**Carcinoma cylindroma** is the name given to a type of cancer where a cylindrical hyaline degeneration has taken place in the center of the cell-nests and the cells are arranged about the degen-

erated area. They occur but rarely in the skin, intestine, and glands.

**Giant-cell carcinoma** is the name given to certain tumors containing very large-sized cells. They are simply hypertrophic, multinuclear cells, or the enlargement may be dropsical.

The black or brown melanin pigment of a **melanocarcinoma** is found partly in the stroma, partly in the cells, giving these tumors a blue-black appearance.

*Seat of Occurrence.*—Such tumors occur in the skin, and always in a normally pigmented area.

**Leukoma**, or **leukoplakia**, is a condition of the tongue which often precedes carcinoma. It appears as one or more white furry patches which are thickened and elevated above the general surface. Numerous deep fissures and ulcers appear from time to time, and later in the disease warts are apt to form. There is a thickening of the epidermis, a growth both upward and downward of the rete mucosum, an elongation downward of the interpapillary cells, and an infiltration or complete obliteration of the papillæ by small round-cells. The condition undoubtedly precedes cancer.

## TERATOMA.

Teratomata are certain tumors presenting a peculiar and complicated growth, and consist of tissues which do not normally occur at the site of the tumor. In a single tumor may be found sometimes fibrillar connective tissue, bone, cartilage, muscle, glands, pigment, etc. There are other tumors that not only have different tissues present, but even more or less complete organs, as skin, hair, teeth or a jaw-bone with teeth, formed pelvic bones, breasts, nerves, muscles, and portions of intestine. These are called **dermoid tumors**, and are apt to be cystic. Such tumors are undoubtedly explained on the supposition of misplaced germinal fragments or that fetal remains have persisted. The latter is certainly true of most of the dermoids, while others probably arise from the inclusion of a twin fetus.

## THE LIVER.

### Normal Structure.

The liver is covered by peritoneum, except for a small area on the posterior surface, and is inclosed in a fibrous capsule. The portal vein, hepatic artery, and bile ducts enter the hilus of the organ surrounded by loose connective tissue (the capsule of Glisson), and these vessels, carried by the connective tissue, make up the framework of the organ. The liver is made up of vast numbers of lobules, which are identical in structure, and it will suffice to describe a single lobule.

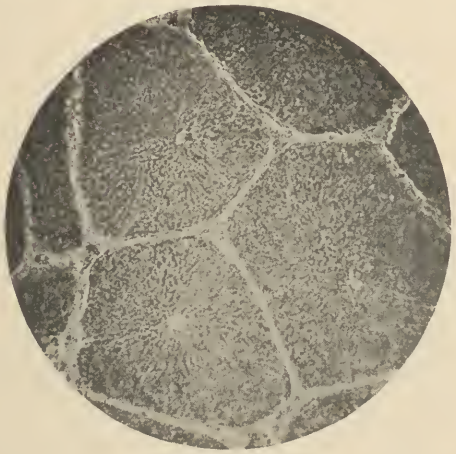

Fig. 48.—Pig's Liver.
Showing the distinct marking of lobules with the interlobular vessels, the central vein, and the capillaries between.

Each **lobule** has an oblong polyhedral shape and is formed by a collection of liver cells arranged around a single terminal radicle of the hepatic vein; hence in cross-section a lobule will always have this **central** or **intralobular vein** in its center. The **liver cells** making up such a lobule are irregular or polygonal in shape, have a granular body, and one or more large oval nuclei. Granules of brown pigment and glycogen and globules of fat are found in the cells. These liver cells are arranged in columns of a single row or many rows, which radiate at right angles from the central vein and are separated from one another by the blood and bile capillaries to be described later.

Now let us follow the three **vessels,** which entered the hilus of the liver, in their divisions and subdivisions until finally we arrive at the lobule we have been describing. These three vessels, now called **interlobular vessels,** will be found on its outside surface, still held together by connective tissue yet ready to break up into minute branches which will cover over the *surface* of the lobule like the net over a balloon. This much will be true of vein, artery, and duct, but from this point each vessel must be considered separately.

The blood from the **interlobular portal vein** is emptied at once into the rich capillary network, which forms an irregular anastomosing series of channels between the columns of liver cells and has a direct course to the *central* or interlobular vein. It is thus evident that the portal blood enters the hilus of the liver, reaches the interlobular veins, then passes through the capillary network around the liver cells directly into the central vein, which is a branch of the hepatic, and from the latter into the vena cava.

The **hepatic artery** carries arterial blood for the nourishment of the elements of the organ. It also breaks up into capillaries on the surface of the lobule, but these capillaries empty into the *portal venous capillaries* at a point about midway between the surface and the center of the lobule, and their blood also passes on into the central vein.

The larger **gall passages** have cylindrical epithelial cells and mucous glands, but in the interlobular bile ducts the glands are lost and the cells are flat or polygonal. At the surface of the lobule these ducts are continuous with the **bile capillaries,** which form a delicate network around each liver cell, yet at all times these capillaries are separated by a portion of the cell diameter from the blood capillaries. They have no walls, but seem to be formed by grooves in the liver cells.

The lobule may be divided pathologically into **three zones:** (1) A peripheral one-third, or portal zone, which is the area of fatty infiltration. (2) The middle one-third, where the capillaries of the hepatic artery empty into the portal capillaries, and which is the area of amyloid degeneration. (3) The central zone around the central vein, or the area of chronic passive hyperemia.

## ACUTE DEGENERATION OF THE LIVER.

Acute degeneration of the liver (*cloudy swelling*) takes place in the acute and infectious fevers, and in certain of the poisons. The liver is swollen, looks boiled, is friable, and the lobules are more distinct. The liver cells are swollen, opaque, filled with albuminous granules, and may become fatty or necrotic, but usually return to normal.

## FATTY INFILTRATION OF THE LIVER.

In fatty infiltration of the liver (*fatty liver*) the cells of the organ, especially in the periphery of the lobule, contain larger or smaller

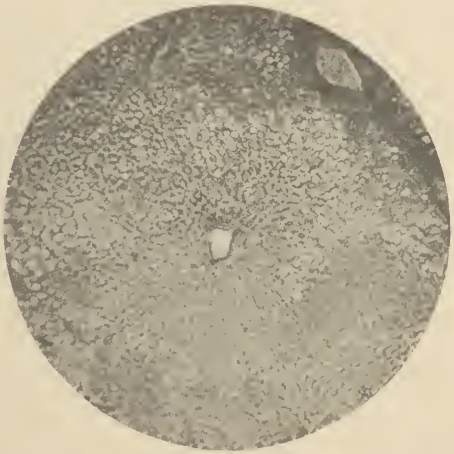

FIG. 49.—FATTY INFILTRATION OF THE LIVER.
The small clear circles represent the fat droplets, and it will be noted that these are in the periphery of the lobule. The central vein is seen in the center.

droplets of fat, even to complete replacement of the cell body by fat, so that the original cell appears as a nucleated crescent at one side of the fat droplet. It most often occurs associated with other lesions, and especially cirrhosis. Such livers are much enlarged, their edges are rounded, the consistency firm; they pit on pressure, are yellowish in color, and the cut surface is greasy. The process may be uniformly distributed through the organ, occur only in patches, or, when it is in the earlier stages, it will be con-

fined to the periphery of the lobules, thus outlining them more distinctly. It occurs in chronic alcohol, phosphorous, and arsenic poisoning, and in conditions of malnutrition and general obesity.

## FATTY DEGENERATION OF THE LIVER.

Fatty degeneration can not always be differentiated from fatty infiltration, but in the former the fat is formed from the protoplasm of the liver cell, the droplets, as a rule, are very small and abundant, the liver cells are angular and clearly in a state of degeneration. It

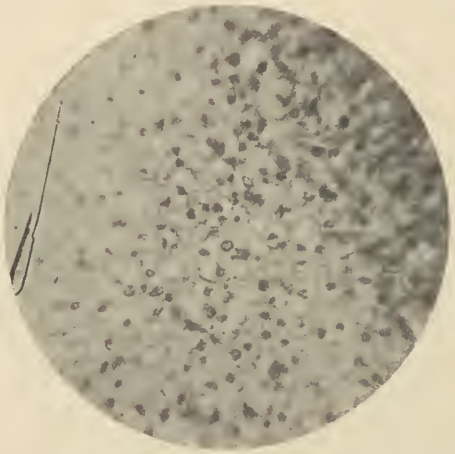

FIG. 50.—FATTY DEGENERATION FOLLOWING PHOSPHOROUS POISONING.

begins in the periphery of the lobule, and usually follows acute degeneration, wasting disease, or acute phosphorous and arsenic poisoning.

## AMYLOID DEGENERATION OF THE LIVER.

An amyloid or waxy liver is one in which an amyloid degeneration, general or local, has taken place in the blood-vessels of the liver. When the amyloid is small in amount, the liver is merely a little firmer; if moderate, translucent spots may be seen, while in extensive degeneration the organ is much increased in size, smooth,

tough, firm, inelastic, and translucent. The liver cells are not invaded by the amyloid material, but they may be much atrophied by pressure. The condition occurs in cachexia, especially of tuberculosis and chronic suppuration.

## CHRONIC PASSIVE HYPEREMIA.

Chronic passive hyperemia of the liver (*nutmeg liver*) occurs in valvular heart disease, pericarditis with effusion, myocarditis, emphysema, pleuritic effusion, and intrathoracic tumors and aneu-

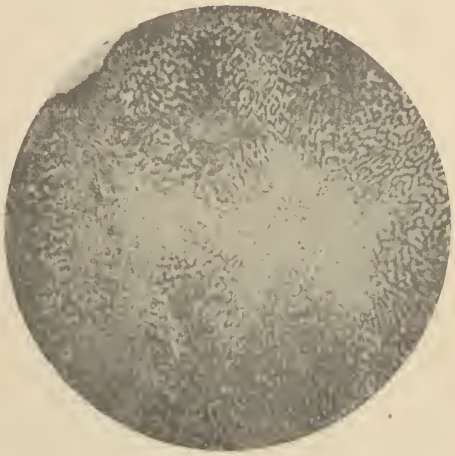

FIG. 51.—CHRONIC PASSIVE HYPEREMIA OF THE LIVER.
In the center of the light-colored area is a central vein, and it will be observed that the liver cells are almost entirely destroyed by the dilated capillaries in this region.

rysms, where the blood-pressure is raised in the vena cava and there is a damming back of the blood in the hepatic and central veins. The capillaries about the center of each lobule are permanently dilated and filled with blood, the liver cells in this region are atrophied and pigmented or have entirely disappeared, the cells of the intermediary zone are tinged yellow by bile, while the cells of the periphery undergo fatty infiltration. The liver is firm and red, mottled like a nutmeg, and normal or diminished in size.

## CIRRHOSIS OF THE LIVER.

Cirrhosis of the liver is a chronic disease characterized by an overgrowth of the connective tissue of the organ and a gradual destruction of the liver cells. This is dependent upon many exciting causes, and, furthermore, it affects the organ differently according to the element in the liver structure which is mainly involved. The following table will include all these variations:

- Cirrhosis.
  - General
    - Primary
      - (*a*) Vascular
        - Venous or common atrophic.
        - Arterial (rare).
      - (*b*) Biliary or Hypertrophic
        - From retention.
        - From radicular angiocolitis.
      - (*c*) Capsular
        - Chronic perihepatitis.
        - Chronic general peritonitis.
      - (*d*) Mixed.
    - Secondary
      - Fatty hypertrophic cirrhosis.
      - Tuberculous cirrhosis.
      - Cardiac cirrhosis.
      - Cirrhosis of Bright's disease.
  - Partial.

## COMMON ATROPHIC CIRRHOSIS OF THE LIVER.

In atrophic cirrhosis the liver is much diminished in size and the surface is roughened (*hobnailed*) by a projection of larger or smaller nodules of liver tissue, which have been pushed out by contraction of the new connective-tissue bands. The cut surface shows a thickened capsule, tough and firm, gray streaks or bands of connective tissue running through the organ, and areas of more or less normal liver tissue between. The general consistency is tough and firm and the liver may be distorted. Microscopically, we find in a **beginning cirrhosis** a moderate increase in the connective tissue of the capsule of Glisson, as it surrounds the medium-sized interlobular vessels; there is a formation of new bile ducts to two, or three, or four, but neither the liver cells nor the vessels seem to be involved. The new connective tissue is primarily rich in round-cells, but as the process advances becomes dense and fibrous, with very few cells. At a somewhat **later stage** of the disease the connective tissue increases in amount, surrounds each lobule or group of lobules, or forms broad, heavy bands through the liver. This new tissue contracts and exerts pressure on the inclosed

lobules, while at the same time it is obstructing or completely obliterating the interlobular veins; less frequently, the bile ducts.

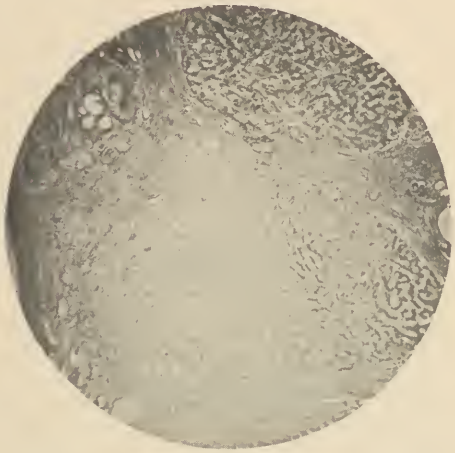

FIG. 52.—ATROPHIC CIRRHOSIS OF THE LIVER.

The liver surface is seen at the left, and from this bands of connective tissue run inward, inclosing areas of liver tissue.

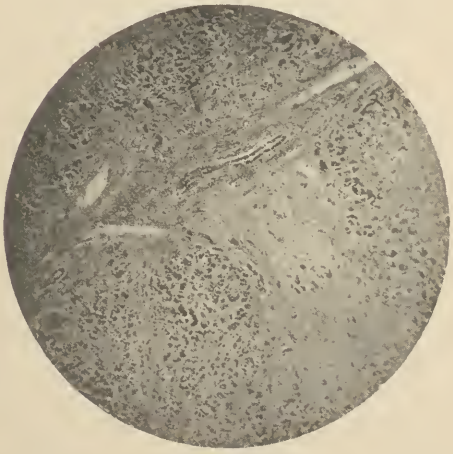

FIG. 53.—ADVANCED ATROPHIC CIRRHOSIS OF THE LIVER.

In the center of the field is a lighter area, which represents the interlobular region, and is one mass of connective tissue. One bile duct is cut longitudinally, and many other newly-formed bile ducts are seen in this area. The border region of the field represents liver lobules invaded by new connective tissue to an extensive degree, and to obliteration of the liver cells.

Now, this combined action of direct pressure upon and interfered nutrition to the cells leads to their atrophy and fatty degeneration.

The connective tissue may extend into the lobules between the cells, cutting off, flattening, finally destroying layer after layer. In this advanced type of cirrhosis there is a very great increase in the new bile ducts and in irregular columns of cuboidal cells representing poorly-formed ducts.

Secondary to the portal obstruction we have ascites, dilatation of the cutaneous veins, hemorrhoids, edema of the feet, enlargement of the spleen, hemorrhage from the stomach and intestines, and a dilatation of the superficial veins of the abdomen, especially about the umbilicus, to form the "caput medusæ."

## HYPERTROPHIC, OR BILIARY, CIRRHOSIS OF THE LIVER.

Hypertrophic cirrhosis is characterized by a very considerable permanent enlargement of the liver to a weight of from seven to ten pounds; the surface is smooth, the consistency exceedingly firm, and it presents, on section, a deep greenish-yellow color. Microscopically, there is a very great increase of connective tissue in the interlobular spaces, and along the capillaries and small bile ducts, between the liver cells. Fibrous bands encroach upon the lobules from all directions, separating the cells or groups of cells. This new tissue resembles in every way that formed in atrophic cirrhosis, but seems to be formed in connection with the *bile ducts* rather than with the portal vein. The bile ducts are greatly increased in number. Despite this extensive and diffuse formation of connective tissue between the cells, however, the nutrition, form, and number of the liver cells is preserved in a remarkable degree as compared with atrophic cirrhosis. In the atrophic form, with limited connective-tissue change, the nutrition of the liver cells is much more seriously interfered with, and the atrophy is out of proportion to the connective-tissue change. Thus, with a diffuse connective-tissue change we get hypertrophy, and with lesser connective-tissue change, yet greater interference with nutrition, we get atrophy. Jaundice is a necessary consequence of the constant and marked involvement of the small bile ducts, and with the absence of portal obstruction ascites does not occur. It is held by many that the new bile ducts are formed by a rapid splitting up of the liver cells, which later go to form flattened cells of these new ducts. The

causes of cirrhosis of the liver are imperfectly understood. In most cases it seems to follow the continued use of strong alcoholic liquors.

## SYPHILITIC HEPATITIS.

This may be distinguished in no way from interstitial hepatitis (*cirrhosis*), except, perhaps, by the heavy bands usually present, or there may be added to the new formation of interstitial tissue

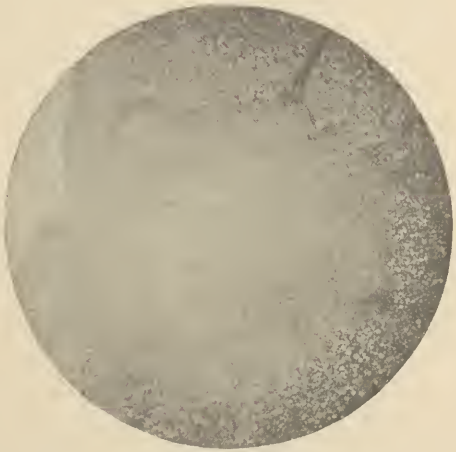

FIG. 54.—MILIARY GUMMA OF THE LIVER OCCURRING IN A CHILD.
The central area is caseous; around this there are small round-cells and connective-tissue fibers, and fibrous bands run out into the liver substance, which is infiltrated by fat.

miliary or very large gummata (see p. 26, Syphilitic Inflammation). Perihepatitis is very commonly a syphilitic process. These forms occur either in the new-born child or in the adult.

## TUBERCULAR HEPATITIS.

Tubercular hepatitis is usually secondary to tuberculosis in other parts of the body, or is a part of a general miliary tuberculosis. Miliary tubercles of varying size are scattered throughout the liver tissue (see p. 21, Tubercular Inflammation).

## ACUTE YELLOW ATROPHY.

In this condition the liver undergoes very rapid diminution in size, to one-half or one-tenth its original volume. The surface is wrinkled, its structure is flabby and soft, and the color is mottled gray, ocher-yellow, or red. The liver cells show every degree of degeneration. In the gray areas the outline of the cells is preserved, but the protoplasm is very granular. The yellow areas may show cell outlines filled with fat droplets and yellow pigment, or there may be complete disintegration of liver tissues, with collections of fat, granules, pigment, and of leucin and tyrosis in place of liver cells. In red areas the cells are entirely absent. The cause of the disease is unknown.

## LEUKOCYTHEMIA (LEUKEMIA) OF THE LIVER.

The liver is enlarged, firm, pale in color, and, on section, the surface is traversed by a network of white tissues, which represent the interlobular spaces. Microscopically, the interlobular spaces are so densely packed with leukocytes that the lobule is distinctly outlined, and the capillaries within the lobule are also crowded with leukocytes, even to atrophy and destruction of the liver cells in places. These cells seem to be emigrated white blood-corpuscles, collected in the tissues without organization. The peripheral zone of the lobule is often pigmented.

## ABSCESS OF THE LIVER.

Abscess of the liver arises from injury, is secondary to inflammation of the gall ducts or surrounding organs, or bacteria are carried by the blood and lymphatic vessels from without. They occur most frequently in the right lobe, are usually single, and are often associated with dysentery, especially of the amebic type. Such an abscess may break externally, or into any adjacent organ or cavity. The abscess contents are purulent, and contain pus-cells, broken down blood and liver cells, pigment, crystals, amebæ, and bacteria. The pus of the amebic form is reddish-brown in appearance and resembles anchovy sauce. The inner layer of the

abscess wall is grayish, ragged, and made up of cloudy or necrotic cells, bacteria, amebæ, etc. The next surrounding row of cells is flattened, somewhat granular, and infiltrated by pus-cells. The

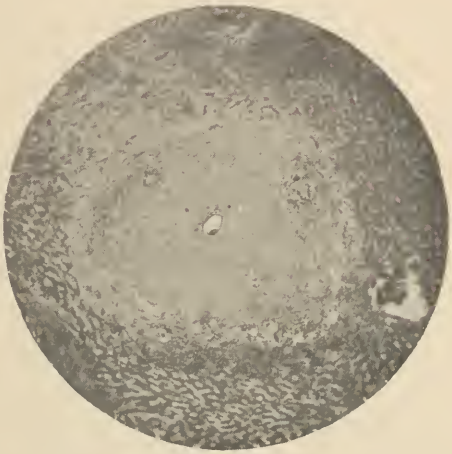

FIG. 55.—MILIARY ABSCESS OF THE LIVER.
The infection in this case seems to have been from the vessel in the center of the abscess.

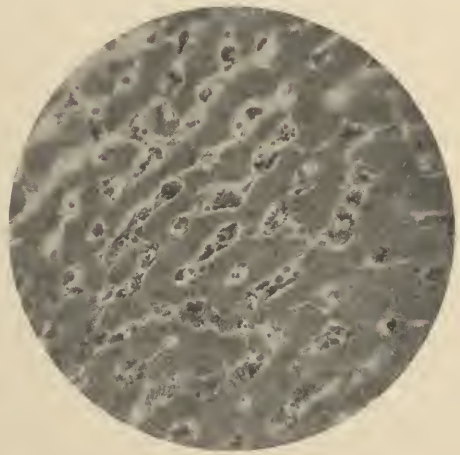

FIG. 56.—PIGMENTED LIVER (BRONZE LIVER) OF MALARIA.
Cells containing pigment are found in the liver capillaries.

outer zone consists of hyperemic liver tissue. Granulation tissue may form on the wall, a fibrous capsule limit the process, and finally a cicatrix represent the site of the abscess. In metastatic

or pyemic abscess there are innumerable minute abscesses throughout the liver.

## PIGMENTATION OF THE LIVER.

Pigmentation of the liver (*bronze liver*) occurs in severe malarial poisoning, where a brown or black melanin pigment, inclosed in variously-shaped cells, is found in the blood-vessels of the liver and sometimes in the cells. This gives a brown or black coloration.

## TUMORS OF THE LIVER.

The **cavernous angiomata** are uncommon tumors of the liver, but may occur in old persons as a single purple patch, ⅓ of an inch in diameter, just beneath the capsule. They are more or less definitely circumscribed by a fibrous capsule, and present a series of communicating cavities, lined by an imperfect layer of endothelial cells, and contain blood.

**Adenomata** may be single or multiple and follow one of two types: (1) They may appear as isolated masses having a structure almost identical with that of normal liver tissue, except that the arrangement is less regular and the cells are larger. (2) The structure may be of irregular or cylindrical cells arranged in more or less tubular form and making up irregular tumors. These are often differentiated with difficulty from carcinoma.

**Carcinomata** develop primarily in the liver from the bile ducts; but of far greater importance are the very common carcinomata which are secondary to primary tumors of the stomach, intestine, uterus, and breast. These secondary growths usually appear as nodules, which are single or multiple, very minute or so large and numerous as to increase the weight of the liver, even to 23 pounds, in the author's experience. They appear as sharply circumscribed, soft or hard masses, white or yellowish in color, and are either embedded in the liver or appear in large numbers projecting from the surface. The liver cells are flattened and atrophied, important vessels may be pressed upon, and the function of the organ is often much impaired. There is another type of *diffuse* infiltration by cancer-cells, in which the cut surface looks mottled and streaked by white carcinomatous bands.

**Echinococcus cysts** of the liver represent a developmental stage of the Tænia echinococcus, and are very frequent in Iceland, Switzerland, Southern Germany, and Australia. They are single or multiple, and vary in size from that of a pinhead to that of a man's head. There is first an outer fibrous capsule, which is formed from the liver, and within this is the true capsule, of parasitic origin, which has a white, opaque, reflecting surface and is distinctly

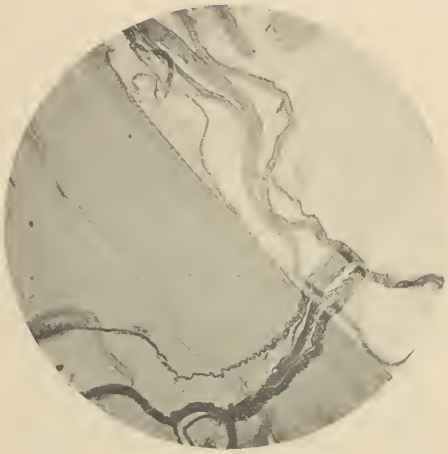

FIG. 57.—WALL OF AN ECHINOCOCCUS CYST OF THE LIVER.
The lamination is well shown.

laminated in structure. From the inner surface of this capsule project the brood capsules and heads of immature tapeworms, as well as secondary and tertiary cysts. The cyst is filled with a non-albuminous fluid containing granular matter, scolices the size of a millet-seed, and hooklets, or there may be blood, pus, fat, cholesterin crystals, bile, etc. The patient may die from the cysts alone, or they may rupture and cause general peritonitis, suppurate and form abscess cavities, or they may heal spontaneously.

# THE RESPIRATORY SYSTEM.

## THE LUNG.

### Normal Structure.

The lungs are made up of a series of conducting tubes and minute cavities, in which air may circulate for oxygen supply to the blood which is so abundantly supplied in the capillary network of their walls. Beginning with the trachea, the bronchi divide regularly

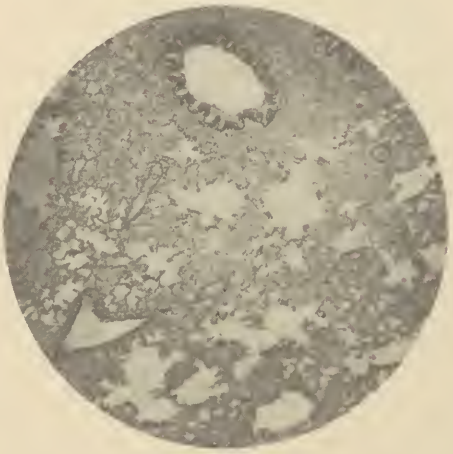

Fig. 58.—Normal Human Lung.

(1) The bronchus to the left with a convoluted mucosa. (2) The clear spaces representing the air-passages and infundibula. (3) The air-vesicles, which are small notches in the walls of these clear spaces. (4) The air-vesicles in the darker parts, which are cut through.

and dichotomously until the terminal bronchus is reached. From such a terminal bronchus numerous short branches or bronchioles are given off, and these very soon open out into the *alveolar* or *air-passages*, which have a very delicate wall made up of a series of small grape-like pouches—the *air-vesicles*. From the air-passage are also given off other larger pouches, which in turn have air-vesicles in their walls, and are called *infundibula*. This terminal bronchiole, with its air-passage, infundibula, and air-vesicles, constitutes a *lobulet*, and a number of these together, corresponding to the single terminal bronchus, constitute a *lobule*. These lobules

are bound together by the interlobular connective-tissue septa to form a *lobe* of the lung. This connective-tissue framework is derived in part from bands sent in from the pleural surface and partly from the connective tissue entering at the root of the lung and running along the bronchi.

The outer connective-tissue coat of the **bronchus** is continuous with the interlobular septa, and forms the fibrous support of the cartilaginous rings. The muscular coat is made up of circular bands of smooth muscle-fiber, and separates the cartilaginous coat from the submucosa, which has a loose areolar connective-tissue structure and carries the blood- and lymph-vessels, and, when present, the racemose mucous glands. A homogeneous basement membrane follows next as we progress inward, and upon this rest three layers of epithelial cells, the innermost or surface layer of which is columnar and ciliated. As the smaller bronchi are reached, the mucous glands and cartilage disappear together, and there is a gradual thinning of the remaining coats.

In the air-passages and air-vesicles the process of thinning is extreme, and the walls consist merely of a striated membranous basement substance containing a few elastic, connective-tissue, and muscle-fibers and lined by epithelium. The alveolar air-passages and bronchioles are lined by two kinds of epithelial cells arranged in a single layer. One type of cell is small, granular, and nucleated, while the other is large, thin, transparent, irregular, without a nucleus, and is called *respiratory epithelium*. Just beneath this epithelium, and separated by it alone from the air-vesicles, lies an abundant network of minute capillaries. Venous blood laden with carbonic acid ($CO_2$), and low in oxygen, is carried by the pulmonary artery to these capillaries. Here, by a process of rapid diffusion, the $CO_2$ is discharged into the air-vesicle, oxygen is taken up from the air contained therein, and the blood returns, bright red and arterial, to the heart by means of the pulmonary veins.

The bronchial artery for the nourishment of the lung has its separate capillary system, and its blood for the most part returns through the bronchial veins, but the two systems seem to anastomose in many places.

## ACUTE BRONCHITIS.

Acute bronchitis is a lesion of the *mucous membrane* of the large and medium-sized bronchi, but the remainder of the lung may be congested. The first stage of inflammation is marked by congestion, redness, and swelling of the mucous membrane, with arrest of the secretion of the mucous glands, and the surface is dry. The mucous glands soon begin to secrete, however, in excessive amount, and a glairy, transparent, tough mucus covers the surface; the ciliated epithelium is almost immediately desquamated, a rapid proliferation sets up in the remaining epithelial cells, the mucous glands are in a condition of cloudy swelling, and leukocytes emigrate in varying amount. There is an expectoration of mucus, epithelial cells, and pus. The smaller bronchi secrete no mucus, because of the absence of mucous glands, but are filled by pus, and may be occluded, with resulting collapse or atelectasis in the area supplied by such bronchus.

## CHRONIC BRONCHITIS.

Chronic bronchitis follows frequent acute attacks, but is usually associated with emphysema, heart disease, interstitial pneumonia, phthisis, etc. The bronchi are congested, dark red in color, smooth and glistening; the mucous glands are enlarged, the epithelium is desquamated and replaced by peg-shaped cells, the connective-tissue coat infiltrated with cells and thickened, and the surface secretes mucus, pus, and round-cells. The blood-vessels may be thickened, the bronchi are often dilated, and the interlobular septa given off from the bronchi may be thickened.

## ACUTE LOBAR PNEUMONIA.

Acute lobar pneumonia is an acute exudative inflammation of one or more lobes of the lung, caused by presence and growth of the Diplococcus lanceolatus of Fränkel, and characterized by a stage of congestion, of exudation, and of resolution.

In the **stage of congestion** the lung is deep red in color, tough, and firm, the cut surface is bathed in blood and serum, but

the lung crepitates, is not solid, and floats in water. The *microscope* shows the capillaries dilated, the alveolar cells swollen or detached, and the alveoli filled with red and white blood-cells, granular

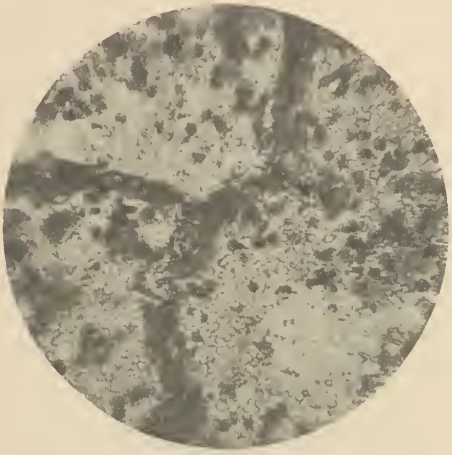

FIG. 59.—ACUTE LOBAR PNEUMONIA. STAGE OF CONGESTION.
The dark bands across the field mark the walls of the acini, which are filled by red blood-corpuscles, pus-cells, and epithelial cells.

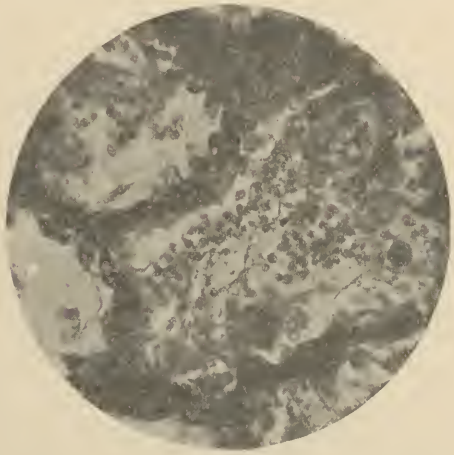

FIG. 60.—ACUTE LOBAR PNEUMONIA. EARLY STAGE.
The acini are filled by many pus-cells, fibrin, and a few epithelial cells. The capillaries in the walls of the alveoli are dilated.

matter, and epithelial cells. The small bronchi contain the same exudate, while the larger bronchi are congested and dry.

In **red hepatization** the lung becomes solid and airless, in-

creased in volume, and very friable; its cut surface is dry, red, and granular, from the filling of the air-spaces. The air-spaces are filled and distended by fibrin, in the meshes of which are pus-cells,

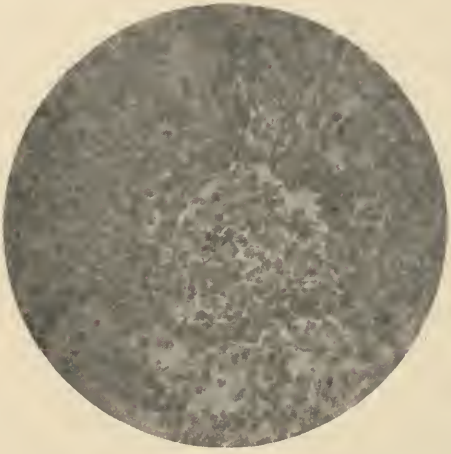

FIG. 61.—ACUTE LOBAR PNEUMONIA. STAGE OF RED HEPATIZATION.
The field is one mass of cells interlaced by fibrin, and the walls of the air-cells are indistinct or faintly visible.

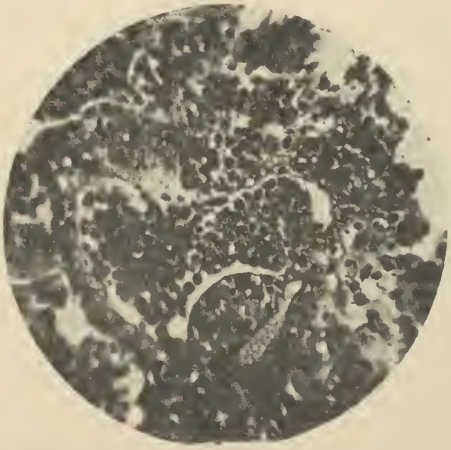

FIG. 62.—ACUTE LOBAR PNEUMONIA. STAGE OF GRAY HEPATIZATION,
Where white blood-cells predominate, and the walls of the acini again become visible.

red blood-cells, epithelial cells, and bacteria. The alveolar wall is infiltrated and indistinct, but despite the pressure the vessels always remain pervious.

In a somewhat later stage, called **gray hepatization,** the red blood-cells have become decolorized and degenerated, the fibrin has disappeared, and the alveoli are densely packed with leukocytes. The exudate begins to soften, and the cut surface is bathed in a purulent fluid. The stage of **resolution** is really a completion of the process of degeneration and absorption already begun, and this stage terminates in return of the lung to normal.

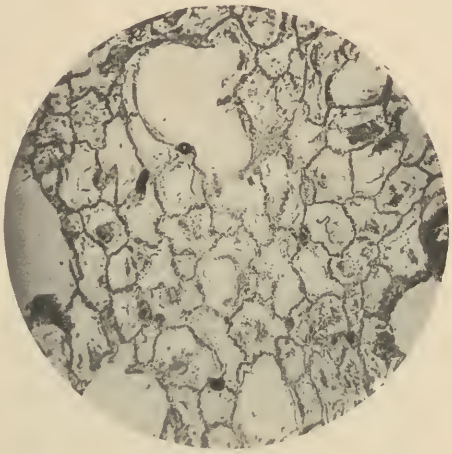

FIG. 63.—ACUTE LOBAR PNEUMONIA. STAGE OF RESOLUTION.
The acini are distinct, and contain fragments of the exudate.

## ACUTE BRONCHOPNEUMONIA.

Acute bronchopneumonia is an inflammation of the **walls** of large numbers of the medium- and small-sized bronchi, and of the lung tissue immediately surrounding these bronchi. It occurs in children as a complication of measles, whooping-cough, and the other infectious diseases, from exposure to cold, and without discoverable cause. It seems necessary that we have, as in lobar pneumonia, an exciting cause for the inflammation, plus the introduction of bacteria, especially the pyogenic cocci, the pneumococcus, the bacillus of Friedländer, or the presence of irritating inorganic substances.

The lung has, on its surface, raised gray areas which are firm and nodular and correspond to the consolidated areas. On section, it drips blood, shows dark red or grayish areas of firm consolidation, corresponding to each bronchus, between which are areas of

edematous and congested lung, which crepitate in most parts, or may show atelectasis from an obstructed bronchus.

*Microscopically*, as pointed out by Dr. Delafield, the walls, and not the mucous membrane, of the bronchi are thickened and infiltrated by cells, while the lumen is filled with leukocytes and epithelial cells. The alveolar walls are much thickened and infiltrated with cells, the air-cells are filled with epithelium and white blood-cells, but fibrin and red blood-cells are not seen. The air-cells immediately about the bronchus are most densely filled with the products of inflammation, while those in the periphery of the consolidated zone are less so, and the air-cells between the areas

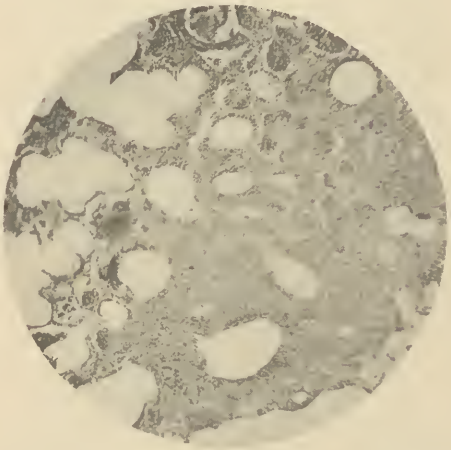

FIG. 64.—ACUTE BRONCHOPNEUMONIA.
The bronchus is seen below with densely infiltrated walls, while the surrounding air-cells are filled by white blood-cells.

have dilated capillaries, swollen cells, and a few cells in the alveolus. It is thus apparent that in bronchopneumonia white blood-cells and epithelial cells predominate in the alveoli, while in lobar pneumonia fibrin predominates. Also that in bronchopneumonia the lung tissue itself (bronchi and alveolar wall) is infiltrated and thickened, making of it an *interstitial* inflammation, while in lobar pneumonia there is merely a *catarrhal* inflammation, or a filling of the *spaces* with inflammatory products.

This difference in the nature of the inflammation explains the long convalescence in bronchopneumonia and the tendency it shows to become chronic. In the **persistent bronchopneumonia**,

the bronchi and alveolar walls become changed into dense connective tissue, the alveoli obliterated, the pleura thickened, other bronchi are dilated, and the entire lung is either studded with fibrous nodules or is made up of one mass of connective tissue. The blood-vessels are not obliterated.

## LOBAR PNEUMONIA WITH THE FORMATION OF INTRA-ALVEOLAR CONNECTIVE TISSUE.

This is a primary inflammation of the lung where, without increase in size, one lobe of the lung is consolidated, smooth, and

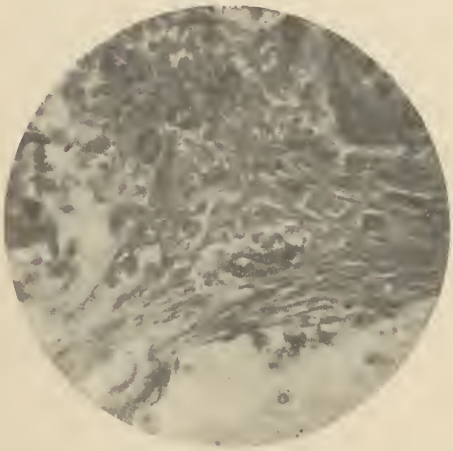

FIG. 65.—PNEUMONIA, WITH THE FORMATION OF INTRA-ALVEOLAR CONNECTIVE TISSUE. The walls of the air-cell are not seen, but the organized plug is well represented.

dense. The alveolar walls are thickened, some of the air-spaces contain fibrin, leukocytes, and epithelial cells, while others, even a few days after the onset of the disease, contain irregular anastomosing plugs of new connective tissue made up of a basement substance and cells. These plugs are definitely connected with the alveolar wall, and a little later contain blood-vessels, which may be injected from the vessels of the lung. The air-spaces are obliterated after a time, and the lung eventually becomes solid and contracted, or portions may break down and form cavities.

## PNEUMONIA OF HEART DISEASE.

The pneumonia of heart disease (*brown induration*) is a chronic inflammation following aortic and mitral disease and affecting both lungs. The lung is decreased in size, dry and leathery in consistency, salmon-pink in color, mottled with brown, and may show hemorrhagic areas. *Microscopically*, (*a*) the capillaries are dilated and so tortuous as to project far into the air-spaces; (*b*) the alveolar wall is much thickened, not alone by the change in the capillaries, but by a new growth of connective tissue and smooth muscle-

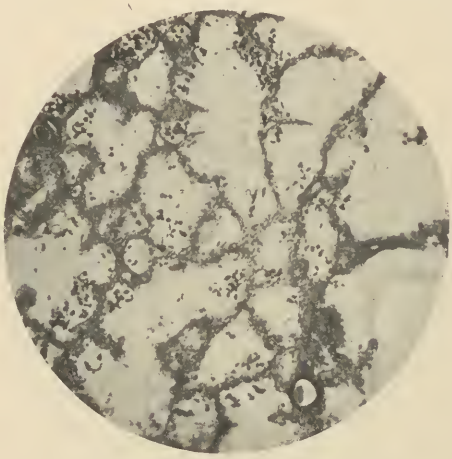

FIG. 66.—PNEUMONIA OF HEART DISEASE.
The walls of the air-cells show dilated tortuous arteries, and a few cells containing brown pigment are seen in each air-cell.

fibers; (*c*) there is a deposition of a brown or black pigment in the alveolar walls and in certain cells found within the alveoli; and (*d*) there is a growth of the cells lining the alveoli.

## INTERSTITIAL PNEUMONIA.

Interstitial pneumonia usually follows some acute inflammation of the lung. If it follows lobar pneumonia, the connective-tissue framework of the lung and the walls of the air-spaces undergo a chronic productive inflammation, with thickening and a result-

ing obliteration of the bronchi and air-spaces. The lobe later becomes small, dense, and firm, and has pleuritic adhesions. When it follows bronchopneumonia, fibrous nodules are formed throughout the lung. Following pleurisy, dense bands run in from the pleura. Following bronchitis and the inhalation of coal- and stone-dust, there are peribronchitic nodules of connective tissue throughout the lung.

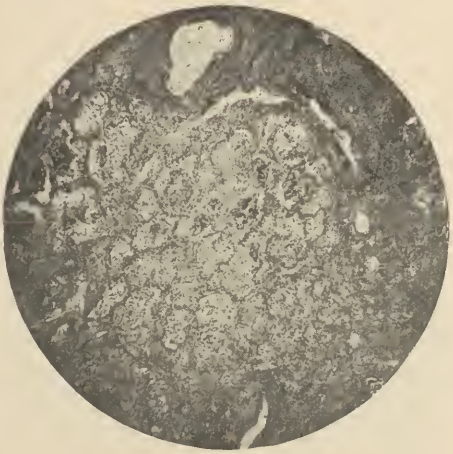

FIG. 67.—INTERSTITIAL PNEUMONIA.

The circumference of the figure shows extensive connective-tissue change, while the alveoli in the center show thickened walls and a lumen filled with cells and organized tissue.

## SYPHILITIC PNEUMONIA.

Syphilitic pneumonia is an interstitial pneumonia, and may occur in any of the forms mentioned under this type of pneumonia. Besides this there may be gummy tumors or areas of necrosis following an obliterating endarteritis.

## TUBERCULAR PNEUMONIA.

Tubercular pneumonia is caused by the lodgment and growth in the lung of tubercle bacilli. These germs may produce: (*a*) an exudative inflammation; (*b*) a productive inflammation, with the growth of new tissue composed of a basement substance and small, round, large, or giant-cells, and called tubercle tissue; or (*c*) they

produce necrosis of the new tissue thus formed. The germs gain entrance to the lung (1) through the bronchi, in which case the involvement is diffuse and associated with an exudative inflammation; or (2) through the vessels, when it is localized and more purely productive.

## ACUTE GENERAL MILIARY TUBERCULOSIS.

In an acute general miliary tuberculosis the lungs are always, and sometimes chiefly, involved, yet there is a general dissemination of

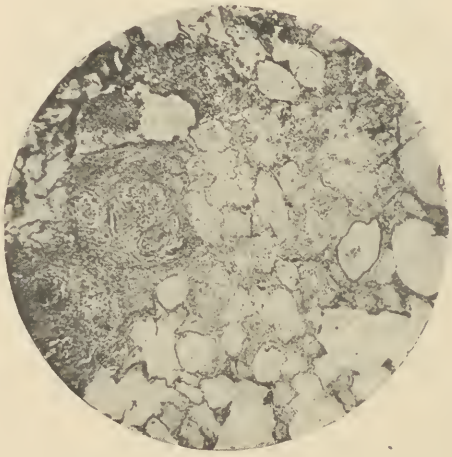

FIG. 68.—ACUTE MILIARY TUBERCULOSIS.

Two miliary tubercles are seen at the left of the diagram, while the surrounding air-cells are filled by the exudate of a simple inflammation.

the tubercular inflammation throughout the body. There is a simple exudative inflammation in all of the lung tissues, with the production of serum, fibrin, and pus. The miliary tubercles are deposited in the walls and cavities of the air-vesicles, in the walls of the bronchi, arteries, and veins, in the connective-tissue septa, and in the pleura. The miliary tubercles may be single and far apart, aggregated, or the lung may be solid by confluence of the groups. (For the characteristics of the miliary tubercle see page 23, Tubercular Inflammation.)

## SUBACUTE MILIARY TUBERCULOSIS.

In subacute miliary tuberculosis of the lung there is less catarrhal inflammation associated with the tuberculosis, but a general or local bronchitis is usually present. The miliary tubercles are confined to the apices, to one lobe, or to a portion of both lungs. They are small in size, usually begin near a bronchus or in an air-space, and are made up of round-cell tubercle tissue. The process may stop at any time and the patient get well.

## CHRONIC MILIARY TUBERCULOSIS.

Chronic miliary tuberculosis begins in the apex of one lung and slowly extends. The miliary tubercles are fewer in number, harder, denser, and more apt to be in a state of cheesy degeneration than the preceding types. In addition to the tubercular inflammation, there is usually either a catarrhal bronchitis, bronchiectasis, an interstitial pneumonia, dilated air-cells, a pleurisy with thickening and adhesion, or a tubercular laryngitis.

## ACUTE PHTHISIS.

In this type of tubercular inflammation there is an acute infection by large numbers of bacilli, but there is also a marked, even predominant, association of an exudative inflammation, caused by simultaneous or subsequent introduction of other bacteria, especially the streptococci. The secondary inflammation renders the consolidation more diffuse, and, most important of all, leads to a very rapid necrosis of the areas of tubercular inflammation. An acute phthisis may affect the lung in different ways: (*a*) There may be a formation of large numbers of miliary tubercles in one or more lobes, and, added to this, the diffuse consolidation of a simple inflammation, thus giving the physical signs and every appearance of lobar pneumonia. (*b*) There may be a tubercular bronchopneumonia, where the small bronchi and surrounding air-spaces are infiltrated and filled with tubercle tissue. This may give the symptoms of a bronchitis, or there may be added a consolidation of the rest of the lung by the products of a simple inflammation.

Again, these areas of tubercular bronchopneumonia may very soon undergo a coagulation necrosis or cheesy degeneration from rapid and early occlusion of the arteries, and give rise to white and yellow

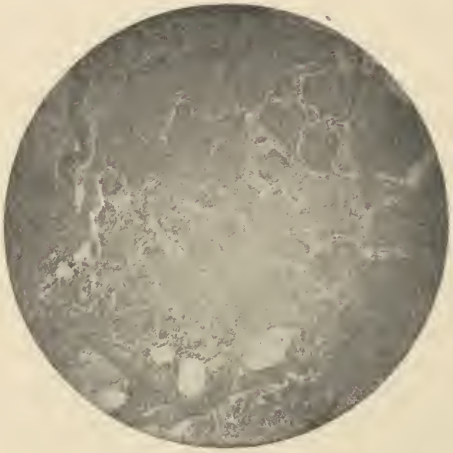

FIG. 69.—ACUTE PHTHISIS.

The air-cells are filled by small round-cells of tubercle tissue, which is already in a condition of cheesy degeneration.

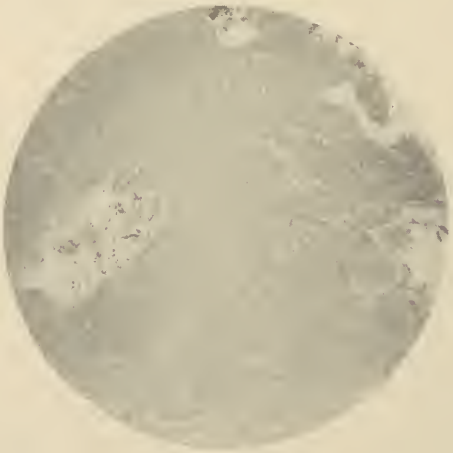

FIG. 70.—ACUTE PHTHISIS, WITH NECROSIS AND CAVITY FORMATION.

areas, which are usually separated by areas of red hepatization. (*c*) The moment that a secondary streptococcus infection reaches these cheesy areas they soften, break down, and form cavities, to

the complete riddling of an entire lobe. (*d*) In addition to the above lesions, bronchiectasiæ may form.

## CHRONIC PHTHISIS.

The lesions of this disease are those of acute phthisis, modified by long continuance. The lung is consolidated and the pleura thickened. The bronchi are the seat of a chronic catarrhal inflammation, and secrete large quantities of mucus and pus; or they are infiltrated by tubercle tissue or miliary tubercles which break

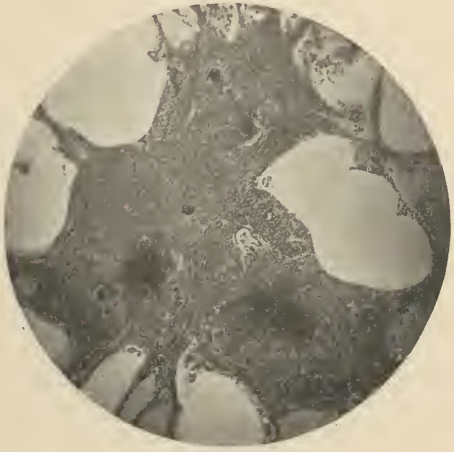

FIG. 71.—CHRONIC PHTHISIS.

Three tubercles are fused together, and present fibrous tissue, small round-cells, giant-cells, and, in the lower two, the dark areas of cheesy degeneration.

down and lead to the formation of bronchiectasiæ. The air-spaces may be (1) partially filled with fibrin, pus, and swollen epithelium and have normal walls; (2) they are tightly packed with tubercle cells and have their vessels occluded; or (3) there may be an intra-alveolar growth of new connective tissue. The tubercular nodules show typical tubercle tissues, or are cheesy, softened, and broken down, or they have been replaced by fibrous tissue and are healed. There may be, furthermore, a general interstitial connective-tissue change. **Cavities** are formed by the dilatation of bronchi which are the seat of tubercular inflammation, or by the breaking down of lung tissue. The walls of such cavities are tubercular and

usually necrotic, and the cavities continue to increase in size by the invasion and destruction of new lung tissue, or by fusion with other cavities, eventually destroying an entire lung. When the process is stationary, they are lined with granulation tissue, which secretes large amounts of pus and mucus; or connective tissue may form in the walls of the cavity, thus limiting the process, and causing "quiescent" or "healed" cavities. Cavities may ulcerate through the pleura, forming pneumo- or pyopneumothorax. The **vessels** usually undergo an obliterating endarteritis as the cavity advances, but they are the last tissues to yield in any necrosis, and fatal *hemorrhage* may result at any time from an artery which is beating free in the cavity.

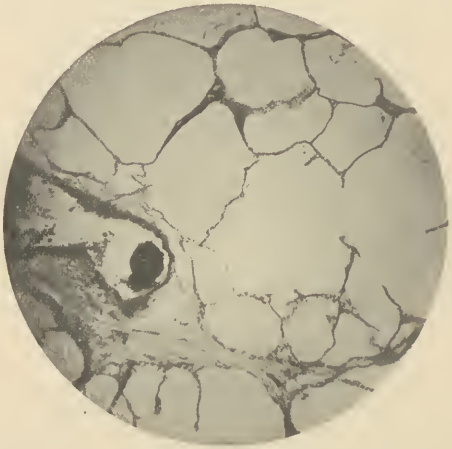

FIG. 72.—EMPHYSEMA OF THE LUNG.

The oblong space at the left represents a bronchus, around which fibrous tissue has formed. The air-cells are much dilated.

## EMPHYSEMA OF THE LUNG.

**Interlobular emphysema** occurs when an air-vesicle or bronchus ruptures and air gains entrance into the intercellular tissue.

**Vesicular emphysema** is **compensatory** when one portion of the lung is disabled and the remaining air-spaces dilate to perform its function; or it is **substantive** when we have a chronic productive inflammation throughout the lung, occurring under the same conditions that determine nephritis, endocarditis, and endarteritis, and characterized by certain clinical symptoms, which are a barrel-shaped chest, certain physical signs, constant and spasmodic

dyspnea, venous congestion of the viscera, changes in the heart, and a tendency to bronchitis. In this last form the lung is increased in size, the bronchi are thickened or thinned, and are usually the seat of a chronic inflammation. The walls of the air-cells are thickened, in many places thinned and dilated, rigid and inelastic; the epithelial cells are increased in size and number; the vessels are pervious. Holes of varying size are found in the walls of many of the air-cells and connect one cell with another. The arteries of the lung are the seat of an endarteritis. Associated lesions are chronic bronchitis, pleurisy, chronic nephritis, endocarditis, and endarteritis.

## ATELECTASIS OF THE LUNG.

Atelectasis is a term given to the collapsed, unaerated condition present in the lung of a child that has never breathed, or in an area of lung supplied by an obstructed bronchus. The lung is solid, non-crepitant, dark blue or purple in color, and smooth on section. The walls of the alveoli are packed together in a structureless mass.

## THE THYROID GLAND.

Colloid degeneration of the thyroid gland is of common occurrence; indeed, when moderate in amount it may be considered as normal. When the degeneration is great it constitutes the condition of goiter, to be described below.

## TUMORS OF THE THYROID.

**Goiter** or **struma** is an enlargement of the thyroid gland. It may be simple hyperemia or **goiter hyperæmica**; but in true goiter there is a hyperplasia throughout the entire gland, giving

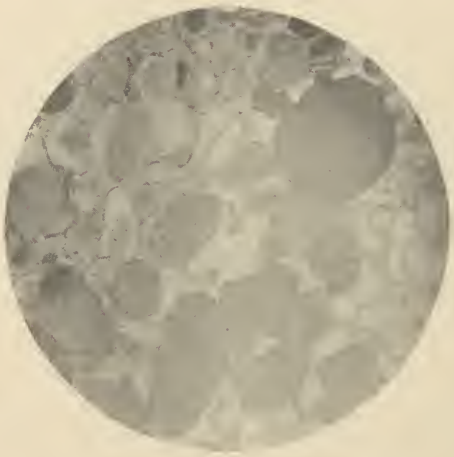

FIG. 73.—COLLOID GOITER.
Vast numbers of acini filled by colloid material make up the tumor.

rise to enlargement. It is a true adenoma, with a growth in the old gland tissue, the formation of new alveoli with a single row of cuboidal epithelium, and a growth of round-cells in the interacinous tissue. There is an associated colloid degeneration, resulting in a collection of colloid material within each acinus to every grade of distention. When the colloid degeneration is extensive, the tumor has a gelatinous appearance, and is called a **colloid goiter**; when large collections of fluid occur in the alveoli it is called **cystic goiter**; and with marked dilatation of the blood-vessels we have a

**telangiectatic struma.** The growth of any goiter is slow, and such tumors are dangerous only from mechanical reasons.

### MYXEDEMA.

Myxedema is a general disease, characterized by a pale elastic swelling of the facial skin, which is rough, dry, waxy, and does not pit on pressure; the hair falls out, the lips are thick, the nostrils broad and thick, and altogether gives a characteristic alteration in the features. There may be similar swellings in other parts of the body. The mental condition is dull, motion is slow, and insanity may result. The subcutaneous tissue has been found in some cases to contain an excess of mucin, but the essential and causative lesion seems to be an atrophy of the thyroid gland, with an entire replacement of gland tissue by fibrillar connective tissue. Endarteritis and chronic nephritis are usually present. Very similar symptoms result from the removal of the thyroid and its destruction by disease, and *cretinism* seems to be very closely allied.

### EXOPHTHALMIC GOITER.

Basedow's or Graves' disease is also a general disease, and is characterized by enlargement of the thyroid, protrusion of the eyeballs, and a rapid pulse. The enlargement of the thyroid is a simple hypertrophy, but is mainly hyperemic, and the left ventricle may be hypertrophied.

## THE SPLEEN.

### Normal Structure.

The spleen is covered by peritoneum, beneath which there is a firm fibrous capsule made up of connective-tissue fibers, elastic fibers, and smooth muscle-cells. At the hilus it runs in as a sheath to the vessels, and this, together with the complicated system of *trabeculæ* sent in from all parts of the capsule, constitutes the supporting framework of the organ.

The artery soon leaves the vein in the spleen and divides into its terminal branches. The walls of such a terminal twig undergo a lymphoid infiltration, until large masses of small round-cells, supported by fine trabeculæ, form eccentrically about it to form the *Malpighian* bodies of the spleen. The splenic pulp is made up of *pulp cords* and *cavernous veins*. The former are innumerable anastomosing cords, made up of a delicate reticular framework, in the meshes of which are small spheroidal lymph-cells, large colorless cells with one or more nuclei, red blood-cells, nucleated red blood-cells, and cells containing pigment. The cavernous veins lie in the

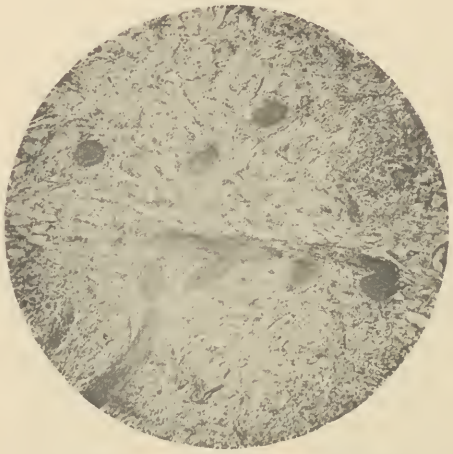

FIG. 74.—CHRONIC CONGESTION OF THE SPLEEN.
There is a growth of fibrous tissue, and the cavernous veins are very definitely dilated.

irregular branching spaces between the pulp cords, and have imperfect or sieve-like walls, made up of elongated and spindle endothelial cells here and there on an equally imperfect fibrous coat.

It will be evident that the arterial blood from the terminal twig of the artery will flow from its capillaries directly into the pulp cords, circulate here among the cells without distinctly walled channels, then percolate through the fenestrated walls of the vein and flow out of the organ. This point is of great pathological importance, as it will explain the frequent acute and chronic blood stagnation in this organ.

## ACTIVE HYPEREMIA OF THE SPLEEN.

Active congestion of the spleen occurs in the acute infectious diseases, such as typhoid fever, pneumonia, diphtheria, etc. The spleen is enlarged, the capsule tense, the pulp soft and dark red. The cavernous veins are dilated and the pulp contains large quantities of red and white blood-cells.

## CHRONIC VENOUS CONGESTION OF THE SPLEEN.

In passive congestion occurring with venous obstruction, there is enlargement of the spleen, dilatation of the cavernous veins, and some thickening of the trabeculæ.

## EMBOLIC INFARCTION OF THE SPLEEN.

Embolic infarctions of the spleen are common. They are red or white according to the age of the infarction, and appear as dark-red, reddish-white, or white circular areas on the surface of the spleen. The entire area of infarction is wedge- or cone-shaped. The red infarct presents little else, microscopically, than a mass of red blood-cells, while the white areas show splenic structure in process of coagulation necrosis, surrounded by an inflammatory area.

## ACUTE SPLENITIS.

Acute splenitis is an inflammatory process which frequently occurs as a secondary lesion accompanying the acute infectious diseases. The spleen is greatly enlarged, its pulp soft, dark- or grayish-red, and the trabeculæ indistinct. There is great hyperemia, plus a swelling and increase in the number of pulp cells. The types of cell are: the large multinuclear, large round, ovoidal, small round (leukocytes), pigmented, or fatty cells, and fragments of red blood-cells. The cells lining the cavernous veins are swollen, and the cells forming the glomeruli sometimes break down and form softened areas resembling abscesses. The spleen returns to normal. The cause of the condition seems to be a lodgment in the spleen of some deleterious materials contained in the blood.

## CHRONIC INDURATIVE SPLENITIS.

Chronic indurative splenitis (*chronic splenic tumor* or *ague cake*) occurs in chronic malarial poisoning, chronic congestion, prolonged typhoid, syphilis, and in leukemia. The spleen is enlarged, the capsule thickened, its consistency firm, and the cut surface grayish-white or deeply pigmented. The hypertrophied trabeculæ are prominent or concealed by the pulp. Microscopically, some of these spleens show a great increase in the size and number of all the cells, with a parallel connective-tissue thickening; while in

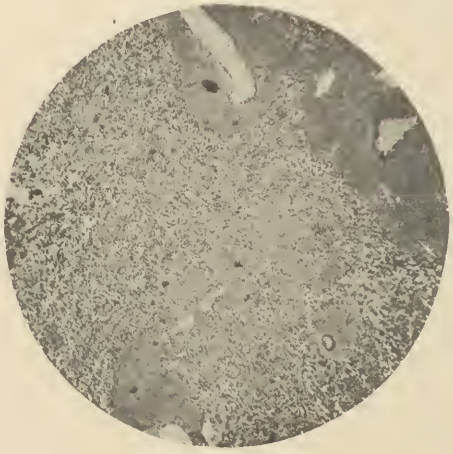

FIG. 75.—CHRONIC INDURATIVE SPLENITIS.

others the connective-tissue thickening is the main change with atrophic changes in the cells. Pigment granules are often deposited in both the cells and the connective tissue.

## SYPHILITIC AND TUBERCULAR SPLENITIS.

Of syphilitic and tubercular splenitis, the same may be said as of the liver.

## LEUKEMIA OF THE SPLEEN.

The spleen of leukemia differs from that of chronic interstitial splenitis in the fact that the enlargement is very much greater, and

the leukocytes are excessive in numbers, to the enormous distention of the sinuses.

## PSEUDOLEUKEMIA OF THE SPLEEN (Hodgkin's Disease).

In this condition the spleen is much enlarged, firm, and tough. The cut surface shows numbers of small, translucent, yellowish masses, corresponding to the Malpighian bodies which have undergone firm fibrous change, and have very few cells. Around these fibrous masses there is a characteristic deposit of golden-brown pigment. The remainder of the spleen is also more fibrous.

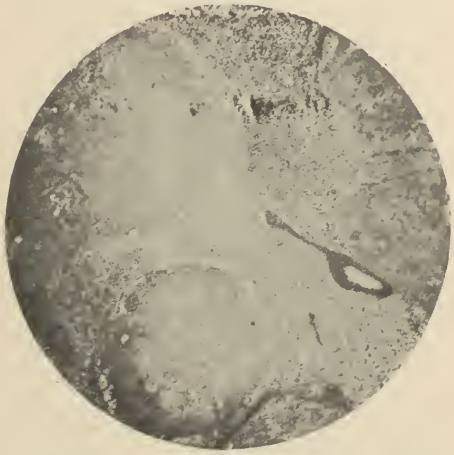

Fig. 76.—Amyloid Degeneration of the Spleen (Sago-spleen).

## WAXY SPLEEN.

Amyloid degeneration of the spleen is usually confined to the walls of the arteries and capillaries, and to the reticulum of the *glomeruli*. The cut surface is sprinkled with translucent areas corresponding to the glomeruli, and resembling boiled sago in appearance. Hence the name "sago-spleen." Another *diffuse* form is also generally described, in which the whole splenic pulp is implicated.

## THE GASTRO-INTESTINAL CANAL.

### THE STOMACH.

The stomach wall is made up of (1) an outer peritoneal covering, (2) a middle muscular structure, consisting of an outer longitudinal, a middle circular, and an inner oblique muscle layer, and (3) an inner lining of mucous membrane, which constitutes its glandular layer. The mucous membrane is attached to the muscular

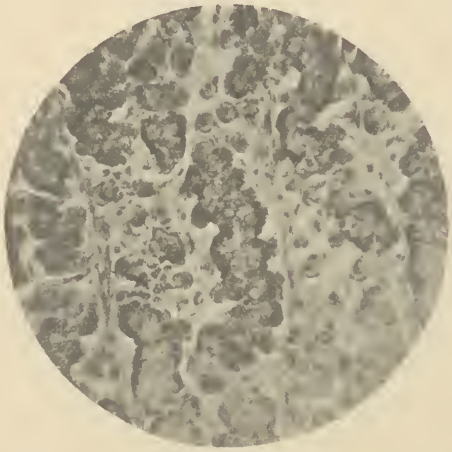

FIG. 77.—NORMAL GLANDS OF THE CARDIAC END OF THE STOMACH.
The outlying parietal cells are well represented.

layer by an areolar submucous tissue, which carries the blood- and lymph-vessels. The *mucous membrane* is entirely made up of simple or divided tubular glands, supported by a small amount of delicate connective tissue, in which are found the blood- and lymph-vessels. These glands are either mucous or peptic, and all have a membrana propria composed of flattened endothelial cells. The entire inner surface of the stomach is covered by columnar epithelium, which also lines completely the mucous glands, but extends only a short distance into the mouths of the peptic glands. The peptic glands are of two kinds: (1) In the *cardiac end*, and, indeed, in the greater part of the stomach, the mouth of the gland is short, the **principal cells** are spheroidal or cuboidal, and either

transparent or finely granular, while outside of these, between them and the membrana propria, are found certain large spheroidal or flattened cells, which are called the **parietal** or **peptic cells.** (2)

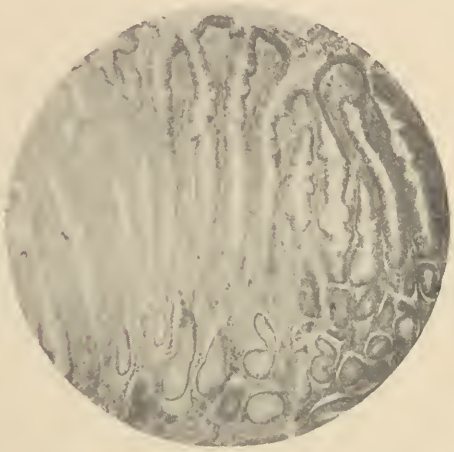

FIG. 78.—NORMAL GLANDS OF THE PYLORIC END OF THE STOMACH.

FIG. 79.—A FOLD OF THE STOMACH MUCOSA WITH THE BLOOD-VESSELS INJECTED.

In the *pyloric end* of the stomach the mouth of the gland is long, the glands are larger and more convoluted, and have *no* large parietal cells. The *arteries* of the stomach and mucosa enter obliquely through the serosa and muscularis, and the distribution

of each arteriole in the mucosa forms a cone, the apex of which is in the submucosa and its base on the surface of the mucous membrane.

### Microscopical Examination of Stomach Contents.

The gastric secretion in fasting shows epithelial cells, mucous corpuscles, cell nuclei, amorphous material, bacteria, and certain snail-shaped bodies formed from mucus. If the normal stomach contents be examined one hour after a test-breakfast, starch granules, plant cells, fat in fine droplets, and bacteria will all be found in small numbers, and muscle-fibers will have lost their striation and be dissociated. Large numbers of starch granules will indicate hyperchlorhydria, while the presence of muscle-fibers that have retained their striation will indicate a diminished gastric juice. It is abundantly proven that micro-organisms may grow in a stomach of normal or high acidity. Among the organisms found are yellow sarcinæ, white yeast, Bacillus subtilis, Staphylococcus cereus albus, etc. Very important among the microscopical findings in pathological conditions are the small pieces of *gastric mucosa* which are almost constantly present in some conditions. They appear as minute red fragments, the size of a pinhead or larger, and, on section, after hardening, embedding, and cutting, will often show the relative changes in the gland and interglandular tissue in such a way that it will be an important aid in diagnosis. Fragments of tumor tissue will, of course, be diagnostic and positive, as in cancer.

## ACUTE GASTRITIS.

In acute gastritis the mucosa is reddened, swollen, secretes less gastric juice, and is covered with mucus, and may show extravasations of blood. The peptic and mucous cells are swollen, granular, and cloudy, and the stroma is infiltrated by leukocytes, or sometimes by small foci of pus.

## TOXIC GASTRITIS.

The ingestion of such poisons as alcohol, phosphorus, arsenic, corrosive sublimate, etc., produces an intense acute gastritis, with swelling, superficial necrosis, hemorrhage, and an acute degeneration

in the gland structures. Poisoning by the mineral acids and strong alkalies produces immediate destruction of the mucosa or other coats, with the formation of a blackened eschar, which is followed by inflammation, sloughing, ulceration, and cicatrization.

## PHLEGMONOUS GASTRITIS.

This is a rare condition, occurring most frequently in men, either idiopathically or as a metastatic process. There is a formation of circumscribed abscesses, or a diffuse, purulent infiltration throughout the submucosa and muscularis, which may be followed by an involvement of the serosa, with perforation and peritonitis. The mucosa is swollen, and may be invaded by the suppurative process.

## CHRONIC GASTRITIS.

Chronic inflammation of the gastric mucous membrane results from dietetic errors, chronic alcoholism, chronic phthisis, Bright's disease, cirrhosis of the liver, heart disease, cancer, etc., and most frequently affects the pyloric end of the stomach. The mucous membrane is congested, slate-gray colored, may show ecchymoses, and is covered by tenacious mucus. It is usually, but not necessarily, thickened, or may present wrinkles and papillary projections, the so-called *état mammeloné*. The submucosa and muscularis are unchanged, thinned, or thickened. *Microscopically*, the *gland-cells* are swollen, granular, and broken down or atrophied, the distinction between the principal and parietal cells is lost, the *tubules* are atrophied and deformed, atypical branches and cysts are formed, and there is a growth of small round-cell tissue in the stroma, pressing the tubules apart. Mucous degeneration in the cells is common and often very extensive.

The result of the prolonged existence of such an inflammation for a long time may be a total atrophy of the glandular coat. This is brought about in two ways: (1) By a fatty degeneration and destruction of the gland tissues, beginning at the surface and extending inward. At a certain stage there will be no glands in the surface, but beneath will still be found glandular cysts, representing the base of former glands, and, still later, glands will be

entirely absent and the mucosa replaced by a small round-cell tissue. Along with this change there is a thinning of the other coats. The condition is sometimes called *phthisis ventriculi.* (2) The process of glandular destruction here begins in the submucosa by a growth of fibrous tissue which is set up by the chronic inflammation. New connective tissue forms between the glands, and constricts and obliterates them until the entire mucosa is replaced by fibrous tissue. The same connective-tissue thickening usually takes place in the walls of the stomach, leading to great thickening and contraction, even to a two-ounce capacity. This is known as *cirrhosis ventriculi.*

## ULCER OF THE STOMACH.

Ulcer of the stomach is a condition where one or more simple ulcers are present in the mucous membrane. They occur most frequently in females, between the ages of twenty and thirty, and are situated in most cases on the posterior wall of the pyloric end at or near the lesser curvature. The typical peptic ulcer is a round or oval funnel-shaped hole in the mucosa, of punched-out appearance, and from ¼ of an inch to six inches in diameter. The surrounding mucosa is not inflamed in the early stages, but later the edges become indurated and elevated. The floor of the ulcer is usually smooth, shows no active inflammatory changes, and is covered with mucus. As the ulcer deepens, the apex of the funnel progressively invades the submucosa, muscularis, and lastly the serosa, to produce perforation and peritonitis or adhesion and fistulous tracts communicating with other viscera or abscess cavities. These fistulous tracts may open into the duodenum, colon, pleura, or pericardium, or the lesser peritoneal cavity may be a huge abscess. Slight or fatal hemorrhages may occur at any time from the erosion of blood-vessels. Healing may be so complete that no trace of a scar is left, but, as a rule, the mucous membrane does not regenerate over the ulcerated area and a dense cicatrix forms a depressed contracted scar. This contraction may cause a stenosis of the pylorus or an hour-glass constriction of the stomach.

The cause of peptic ulcer is not well understood. It is undoubtedly due to interfered nutrition in circumscribed areas, followed by a *digestion* and destruction of this area by gastric juice,

but the cause of such obliteration in the arteries is not known. *Spasm* of the arteries, *obliterating endarteritis*, *thrombosis*, and *embolism*, have each their advocates as probable explanations.

## TUMORS OF THE STOMACH.

**Papillomata** occur frequently in chronic gastritis, as previously stated, in the form of polypoid hypertrophies. They also occur as independent polypi, having a connective-tissue stroma arranged in tufts and covered by columnar epithelium.

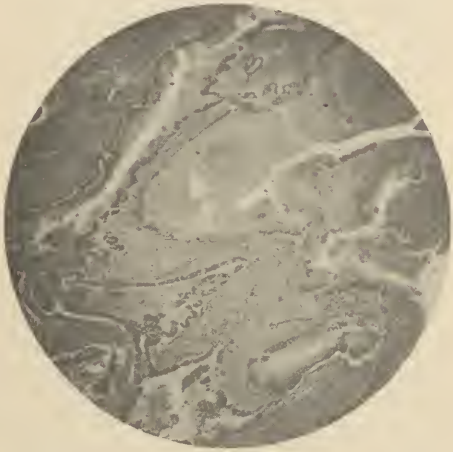

FIG. 80.—MALIGNANT ADENOMA (ADENOMA DESTRUENS) OF THE STOMACH.
The acini are very irregular, but lined everywhere by a single row of columnar epithelium.

**Fibromata, myomata, adenomata,** and **sarcomata** are of rare occurrence.

**Carcinoma** is very frequent, and is of great importance as a lesion of the stomach. It is almost always primary, and has its origin in the mucosa, from which it may grow to infiltrate all of the coats and the surrounding organs.

The varieties which are found are: (1) *Cylindrical-cell* or *adeno-carcinoma*, which form soft, nodular tumors on the surface of the mucous membrane, and are characterized by the predominance of tubules lined with cylindrical epithelial cells, resembling those of normal glands. As the growth develops, the regular tubules are

lost by a filling of the spaces by cells. (2) *Medullary carcinomata* are characterized by large, flat, soft fungating masses projecting above the mucosa. They are yellowish-white in color, made up of cells and vessels, with very little stroma, are very subject to hemorrhage, degeneration, and ulceration, and form early metastases. (3) The *scirrhous carcinomata* of the stomach are almost cartilaginous at times in their firmness, have abundant connective tissue, few cells, do not ulcerate, and rarely form metastases. (4) *Colloid carcinoma* occurs when there is a colloid degeneration in the first and second varieties, and the tumor has a gelatinous, transparent appearance.

Sixty per cent. of carcinomata occur in the pyloric end of the stomach, and the lesser curvature and posterior wall come next in frequency. Some of these carcinomata form large tumors, projecting into the stomach; others form annular rings about the pylorus, producing stenosis; or the growth will spring from the deeper layers of the mucosa, form a circumscribed, flat tumor, which first lifts up the mucosa, then ulcerates, and the tumor extends in all directions. Such ulcers keep pace with the tumor growth until they reach a large size and perforate or involve surrounding tissue. Lastly, the growth may be from the deep layers of the mucosa, and extends along the plane of the submucosa to cover large areas, but forms no tumor and does not ulcerate.

Metastatic growths occur in 48 per cent. of cases, and 40 per cent. are in the liver, the infection taking place through the portal circulation. The lymphatics are also infected sometimes. The mucous membrane of the stomach, as a rule, presents the characteristic changes of a chronic gastritis.

## THE SMALL INTESTINE.

### Normal Structure.

The coats of the intestine are the same as those of the stomach, but the mucosa presents marked differences. This is thrown into many crescentic valve-like folds, called *valvulæ conniventes*, and its entire surface is covered by conical or finger-like projections, called *villi*, in the center of which are the large lymph- or chyle-spaces, muscle-fibers, and loops of blood-vessels. Between and at the

base of these villi are simple tubular glands, called the *follicles* or *crypts of Lieberkühn*. Covering the villi and lining these glands is a single continuous layer of columnar epithelial cells, with more

FIG. 81.—NORMAL SMALL INTESTINE.

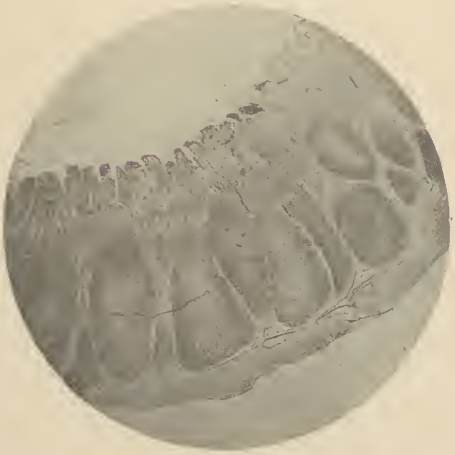

FIG. 82.—PEYER'S PATCH FROM THE INTESTINE OF A DOG.
The separate collections of lymphoid tissue surrounded by fibrous septa is well demonstrated.

or less abundant, clear, ovoidal, goblet, or mucus-secreting cells scattered here and there between them. The *muscularis mucosæ* has an importance in the intestine because muscle-fibers run from

it into the villi. In addition to the glands mentioned, there are certain racemose, mucous glands in the duodenum, known as *Brunner's glands.*

Most important of all, perhaps, from a pathological standpoint, are the *solitary* and *agminated nodules*, or *Peyer's patches.* (1) The solitary nodules are found throughout the small and large intestine as small grayish nodules which lie in the mucosa, often extending beneath the muscularis mucosæ, and project into the lumen of the intestine. Their structure is of simple adenoid tissue, such as is found in lymph-glands, and is that of delicate trabeculæ filled by small round-cells. (2) The Peyer's patch is found only in the small intestine, and for the most part in the lower part of the jejunum and in the ileum. They are round or oval projections which are always on the side opposite the mesenteric attachment, and their structure is that of an aggregation of solitary nodules.

### Acute Catarrhal Enteritis.

An acute catarrhal inflammation of the intestine arises idiopathically, as a result of dietetic errors or toxic materials, in cholera morbus, etc. There may be no visible change in the mucosa, even though the symptoms have been severe; or there will be present swelling and infiltration of the mucosa with degeneration and exfoliation of the epithelial cells, and a coating of mucus. The solitary and agminated follicles are usually swollen, especially in children.

### Typhoid Fever.

In typhoid fever there is more or less general catarrhal inflammation of the small intestine, but the characteristic lesions of the disease are confined to the lower end of the ileum, and consist in a *hyperplasia*, *breaking down*, and *ulceration* of the solitary and agminated follicles in this part of the intestine. In the early stages the mucosa is congested and covered by minute, or large, round, and oval projections which may be so large as to occlude the intestine. Later each nodule, but, as a rule, a very few, may be replaced by a superficial or deep ulcer. The changes taking place may be divided into four stages. In the **stage of hyperplasia** the patches are enlarged in varying degrees; pink, semi-transparent, and lighter in color than the surrounding mucosa; soft and spongy in consistency. The surface of a Peyer's patch presents a ridged or

convoluted appearance, owing to the fact that the lymph nodules have swollen while the septa between them have remained unchanged. Microscopically, there is intense hyperemia of the

FIG. 83.—TYPHOID ULCER WITH THE INFILTRATED SUBMUCOSA AS ITS FLOOR.
One edge of the ulcer is seen at the right with the overhanging edge turned upward. The other edge is barely seen at the left.

FIG. 84.—PERFORATING TYPHOID ULCER.
A portion of the floor of the ulcer at the point of perforation is shown.

nodules, with an excessive production of small round-cells, resembling lymph-cells, of large round- or multinuclear cells, and sometimes of red blood-cells. The new cells are not confined to

the follicles, but infiltrate the mucous, muscular, subserous, and serous coats. The condition may go no further, and resolution take place with gradual disintegration and absorption of the cells, *or*, with the excessive accumulation of the cells, the vessels are occluded, and we have the **stage of necrosis.** A part or the entire nodule becomes white and gangrenous, and a slough is formed involving the surface of the mucosa, the entire mucosa, or more frequently it extends through the submucous coat to the muscularis. In other cases where perforation occurs, the slough invades the muscularis and serosa. **Ulceration** follows the separation of these sloughs, when ulcers are formed with swollen

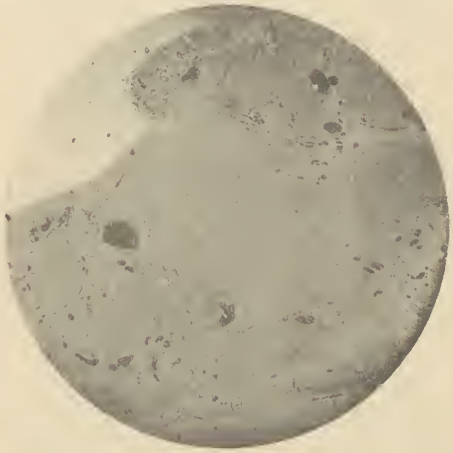

FIG. 85.—TUBERCULAR ULCER OF THE INTESTINE.

The mucosa is seen above, but the entire thickness of the intestinal wall to the peritoneal surface below, consists of tubercular nodules.

overhanging edges, which are infiltrated with cells. The floor of the ulcer is usually formed by the infiltrated muscularis. The shape and size of the ulcers depend upon the amount of sloughing that has taken place, because an entire Peyer's patch does not always break down, but round and oval ulcers are the rule. Hemorrhage and perforation take place with the separation of the slough. The cell growth continues, and the ulcer increases in size, or **healing** begins. In the process of healing the edges fall down and unite with the floor, granulation tissue forms, and the glandular elements are reformed by an extension growth from the edges. A characteristic, smooth, grayish, depressed spot is left.

### Tubercular Inflammation.

Tubercular inflammation of the small intestine may be primary or it may be secondary to tuberculosis of the lung or peritoneum, and affects mainly the solitary and agminated lymph-nodes of the intestine. Tubercular nodules form, rapidly undergo cheesy degeneration, slough, and leave deep, ragged ulcers with infiltrated overhanging edges. These ulcers usually extend through the muscular coats. The floor of the ulcer is rough and nodular, and often shows the invasion of the subserous lymphatics by tubercle nodules which appear in the floor of the ulcer as yellow, star-shaped markings. The ulcer is oval, with its long axis transverse, and extends laterally oftentimes to completely surround the bowel. The peritoneal surface corresponding to the floor of the ulcer shows shot-like nodules and the same star-shaped infiltration of the lymphatics. Such ulcers rarely perforate because of the progressive proliferation and thickening taking place in their floor, which causes thickening and adhesions rather than perforation. They show very little tendency to heal, and leave puckered cicatrices when they do. To differentiate tubercular from typhoid ulcers the following table may be used:

| | Typhoid Ulcer. | Tubercular Ulcer. |
|---|---|---|
| Long Axis. | Longitudinal. | Transverse. |
| Edges. | Ragged, float out in water, thin, vascular, and undermined. | Thick, prominent, nodular, not undermined, terraced or sloping, pale or red, and made up of tubercle tissue. |
| Floor. | Smooth and vascular. | Nodular, irregular, and streaked with yellow. |
| Peritoneum. | Smooth or, perhaps, perforated. | Thickened, yellow or gray points running along lymphatics. |
| Perforation. | Common. | Very rare. |
| Peritonitis. | Common. | Rare. |
| Hemorrhage. | Common. | Less frequent. |
| Extension. | Longitudinally or in depth. | Transversely. |
| Healing. | Prompt, with small cicatrix. | Rarely heal, or, form puckered cicatrices. |
| Bacteria. | Typhoid bacillus (may be found). | Tubercle bacillus. |

## THE LARGE INTESTINE.

The mucous membrane of the large intestine is thickly set with tubular glands somewhat larger than those found in the small intestine, and there are many solitary nodules. The villi and Peyer's patches, however, are absent.

### Acute Catarrhal Colitis.

Acute catarrhal colitis is a simple exudative inflammation of the colon in which there is congestion and swelling of the mucosa, especially on the summits of folds, an infiltration with serum and

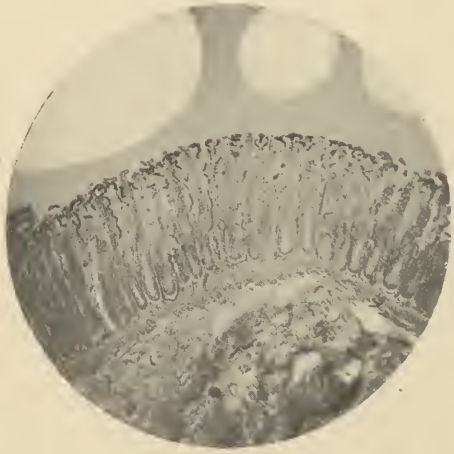

Fig. 86.—Normal Large Intestine.

pus, a mucopurulent exudate on the surface of the mucous membrane, and, when severe, minute superficial ulcers may form. In certain severe forms of the disease pus-cells infiltrate the walls of the colon in excessive numbers, and the patients die in a very few days. Added to the catarrhal inflammation, there may be from the start a formation between the glands of new connective tissue, rich in cells, which causes thickening of the colon and results in a chronic colitis.

### Chronic Colitis.

Chronic colitis, then, is a condition where there is a general infiltration by connective tissue of the mucosa or of all the coats of the colon. The inner surface of the colon is rough, irregular, or poly-

poid, gray or blackish in color, and ulcers in all stages are usually found. The caliber of the bowel is reduced.

### Croupous Colitis.

In croupous colitis there is a croupous inflammation of the mucous membrane of the colon. There is a congestion, swelling, and deep infiltration of all the coats with serum and pus, and there is a coagulation necrosis in the exudate on the surface of the mucous membrane giving rise to a false membrane.

The false membrane is usually deposited in patches on the tops

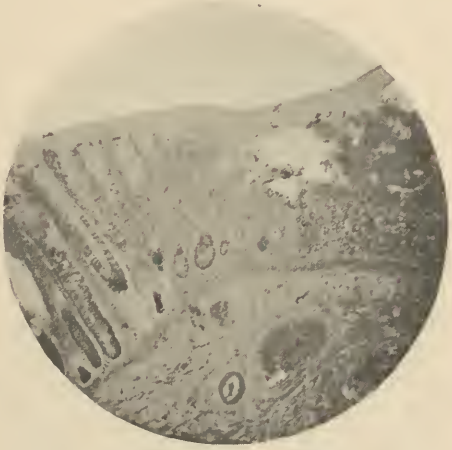

Fig. 87.—Ulcerative Colitis.

A few glands in a condition of mucous degeneration are seen at the left, and the necrotic and broken-down mucosa to the right.

of folds or it may be general. The intestine may return to the normal, or there may be a necrosis of tissue to almost any depth, which will be followed by ulceration and a chronic colitis with unhealed ulcers, or cicatrization may take place.

### Follicular Colitis.

In follicular or *nodular* colitis there is a catarrhal injection and swelling of the mucous membrane of the colon, but the most prominent part of the lesion seems to be an inflammation of the solitary follicles, which are congested, swollen, and stand out prominently. They very soon necrose and leave round ulcers

with overhanging edges, which show no tendency to heal. The stools are diarrheal rather than dysenteric.

### Amebic Colitis.

Amebic colitis is an inflammation of the colon caused by the presence and growth of the *Amœba coli*. This is a unicellular, protoplasmic, motile organism, 15 to 30 micromillimeters in diameter, made up of a clear outer zone and a granular inner zone, inclosing a nucleus and vacuoles. These amebæ enter the tissues and produce a general or local infiltration. The *general* infiltration is due to an edema, to a multiplication of the connective-tissue cells, and

Fig. 88.—Acute Nodular Colitis.

A solitary nodule is seen in the process of disintegration, and a deep ulcer will soon form at this point.

to an infiltration by pus-cells and amebæ of the submucosa and intertubular connective tissue. The effect of the presence of the Amœba coli seems to be to cause death of tissue, and we find either circumscribed areas of necrosis in the mucosa giving rise to superficial ulcers, or a very extensive necrosis of all the layers giving rise to very large and deep ulcers. The whole process, however, may be *localized* in spots throughout the colon. In this instance the submucosa is first involved, and a nodule forms by a local multiplication of connective-tissue cells. The mucosa necroses only in a limited area, while the necrosis is extensive in the submucosa, where the greater part of the nodule lies, and deep, irregular,

undermined ulcers are formed with overhanging edges and a yellow gelatinous floor. Ulcers in this way undermine the mucosa and unite with others until the whole colon may be riddled with sinuses beneath a more or less normal mucosa, presenting ulcers only in places. These ulcers may invade all the coats, and whole areas of mucosa or muscularis may slough *en masse* in advanced stages of the disease. The amebæ are found in the floor of the ulcers, and in the connective-tissue coat as little white gelatinous masses.

### Necrotic Colitis.

Necrotic colitis is an uncommon and very fatal form of inflammation of the colon where there is rapid and extensive infiltration and necrosis of the submucosa with the formation of undermining ulcers, leaving the mucosa separated over large areas from the muscularis.

Fig. 89.—Acute Catarrhal Appendicitis.

From a man aged thirty-two, sick two days, temperature 102° F., pulse, 110. There is great thickening and infiltration of the mucosa, but no thickening of the muscularis or serosa.

### The Vermiform Appendix.

The vermiform appendix may be the seat of a variety of inflammatory conditions. (*a*) The *mucosa* may be the seat of a catarrhal inflammation. (*b*) The *entire wall* may be the seat of an acute exudative inflammation. There is no necrosis or perforation, but the wall of the appendix is infiltrated and thickened even to one inch in

diameter, and, if free in the peritoneal cavity, will set up general peritonitis. (*c*) In one or more places the entire thickness of the wall of the appendix is the seat of a *necrotic* inflammation with sloughing of the tissue, perforation into the peritoneal cavity, the discharge of the fecal concretion, which is usually contained, and

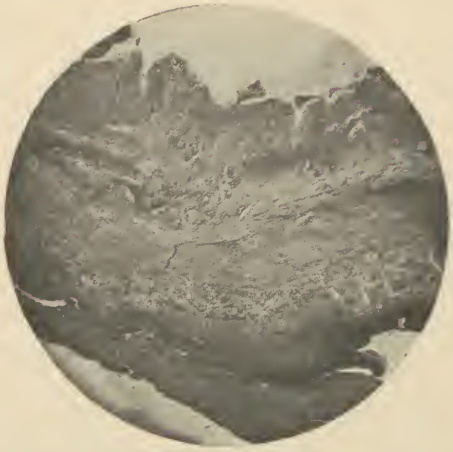

FIG. 90.—SUPPURATIVE APPENDICITIS WITH NECROSIS AND PERFORATION.
All of the coats are thickened and infiltrated by pus-cells. The mucosa is above, the serosa below.

the production of a general peritonitis or localized abscess. (*d*) The entire appendix may become *gangrenous* at once and set up severe general or local peritonitis.

## HEART.

### ACUTE DEGENERATION.

Cloudy swelling, or acute degeneration, of the heart is a condition taking place in the acute infectious fevers. The heart muscle is of a uniform grayish color, and the consistency is soft and friable. The muscle-fibers are filled by minute albuminous granules, which often render the fibers opaque and obscure the striations and nuclei. The granules are less refractile than fat, and do not dissolve in ether or stain with osmic acid, but are soluble in acetic acid.

## FATTY DEGENERATION.

Fatty degeneration is a condition present in a large variety of general and local diseases, follows the ingestion of such poisons as phosphorus, arsenic, and antimony, and occurs in diphtheria. Prominent among the causes is obliteration or atheroma of the coronary arteries. The degeneration is uniform or in patches, giving the muscle a pale-yellow color and flabby consistency. The muscle-fibers are infiltrated and filled with small and large droplets of fat, often to obliteration of their markings. The globules present are larger than in cloudy swelling, stain with osmic acid, are more

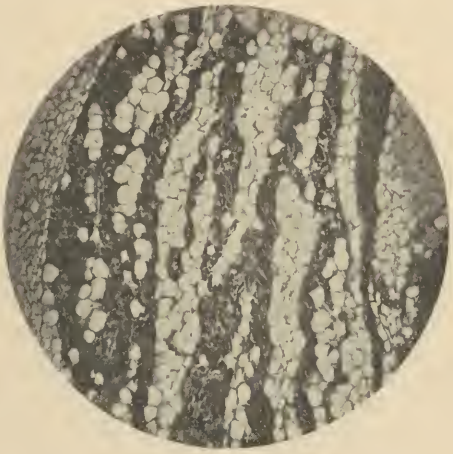

FIG. 91.—FATTY INFILTRATION OF THE HEART.
The dark bands represent heart muscle, and the clear circles fat droplets.

refractile, and are soluble in ether. Fatty degeneration leads to thinning and weakening of the heart walls, and sometimes causes sudden death by heart rupture or heart failure, or aneurysm of the heart may also be formed, and precede a rupture.

## FATTY INFILTRATION.

Fat infiltration, or lipomatosis, is a condition distinct from the above, where fat is deposited about the heart and between its fibers. It occurs in obesity and in debilitated persons.

## BROWN ATROPHY.

The heart, in this condition, is dark brown in color, and diminished in size. The walls are thinned, firm, and tough or brittle, and in the center of each muscle-fiber is a dark-brown spot. These brown spots are made up of a finely-granular pigment which obscures the nucleus. The pigment seems to be derived from hemoglobin.

## CORONARY ARTERIES.

*Sudden obliteration* of the coronary arteries, by an embolus, is a common cause of sudden death, or it may cause fatty degeneration

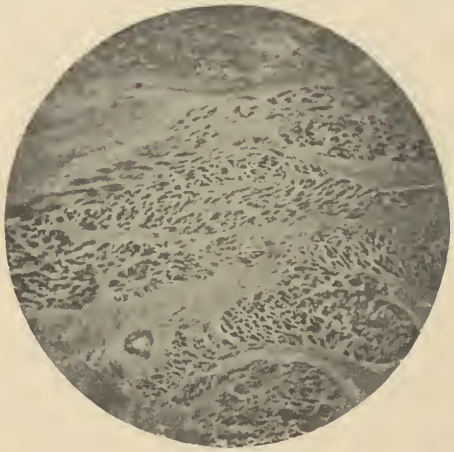

FIG. 92.—INTERSTITIAL MYOCARDITIS FOLLOWING ARTERIOSCLEROSIS OF THE ANTERIOR CORONARY ARTERY.

of the heart muscle, or anemic necrosis, with rapid breaking down. When there is a slow obliteration of these arteries, a condition of **chronic interstitial myocarditis** is produced. In this condition there is a growth of connective tissue between and around the muscle-fibers, leading to their atrophy and obliteration, with resulting dilatation, aneurysm, or thrombosis of the heart cavity.

## ACUTE PURULENT MYOCARDITIS.

Acute purulent myocarditis may occur in scarlatina, in pyemia, the acute infectious diseases, etc. It may be a diffuse infiltration

by pus-cells between the muscle-fibers, or there may be circumscribed abscesses, which will weaken the heart wall and lead to its rupture.

## ACUTE ENDOCARDITIS.

In simple acute endocarditis, occurring in rheumatism, the valves of the heart are thickened and swollen by the formation of a very cellular new connective tissue beneath the endocardium. They are succulent and smooth, or their contact surface is roughened by the presence of fibrin, or by soft, friable, translucent

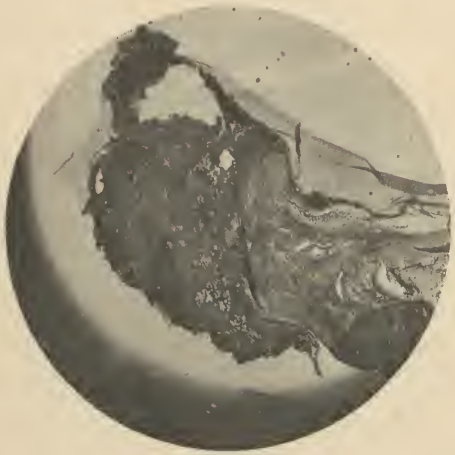

FIG. 93.—VEGETATION ON MITRAL VALVE IN A CASE OF ACUTE ENDOCARDITIS.
The darker portion at the left represents the vegetation adherent to the tip of the mitral valve.

fungosities, called vegetations. The proliferating connective tissue formed beneath the epithelium breaks through and forms a surface of granulation tissue made up of loops of newly-formed budding blood-vessels. To this granulation surface is firmly attached the vegetation, which is mainly made up of a network of fibrin, into which the vessels from beneath are forcing their way. There are very few cells in the structure of such a vegetation, but the compressed layer of fibrin on the surface, which is in contact with the blood, contains large numbers of leukocytes in its meshes.

When the inflammatory process is intense, the valves may lose their endothelium in places, or there may be separation of vege-

tations, and ulcers are formed. When bacteria are present in an acute endocarditis, the type known as **malignant endocarditis** is present, and the old and new tissue proliferates to form extensive organized vegetations on the valves and endocardium of the cavities. Bacteria are present in these vegetations, and cause necrosis with deep ulceration, while detached vegetations form infectious emboli, which cause abscesses in various remote parts of the body and lead to pyemia.

## CHRONIC ENDOCARDITIS.

In chronic endocarditis the aortic and mitral valves are most frequently affected. The valves, in most cases, have smooth sur-

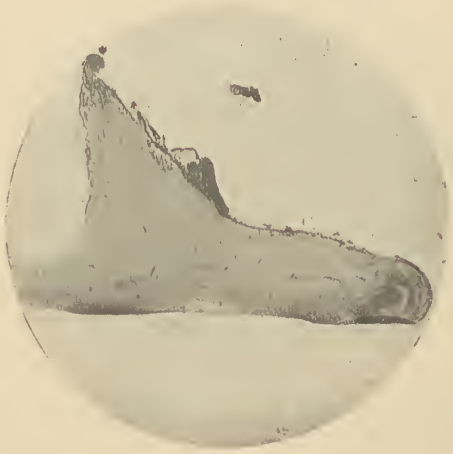

FIG. 94.—VEGETATION ON HEART VALVE IN CHRONIC ENDOCARDITIS.
The vegetation which projects upward consists of rather dense fibrous tissue. A small blood-clot is adherent at the right.

faces and thickened edges, or they may be dense, contracted, and infiltrated by the salts of lime. In the milder forms the transparency of the valves is lost; small nodules appear on their borders, or a grayish-red swelling forms just within the border at the point of maximum contact. As connective-tissue growth continues, the edges become twisted, curled, and the angles of the valves grow together, narrowing the orifice. Necrosis occurs in some places, and growth of vegetations in others, together with a contraction of

the new fibrous tissue and a shortening of the chordæ tendineæ. The important result of all these changes is the destruction of the accurate, even at times possible, apposition of the valves, and a resulting insufficiency, or stenosis, or both. This process of thickening goes on to an advanced degree before the lime salts are deposited. In a chronic endocarditis every grade of involvement is included, from the merest thickening to total destruction and calcification.

## THE ARTERIES.

### Normal Structure.

The structure of the arteries will necessarily vary with their size. The **capillaries** are simple endothelial tubes, made up of a single row of thin, flat endothelial cells with a single nucleus, cemented together by their edges.

In the **smaller arteries** there are three distinct coats: the intima, media, and adventitia. The *intima* consists (1) of a layer of endothelial cells continuous with those forming the capillary wall, (2) an intermediary layer made up of large branching cells and of fibrillated and elastic fibers, and (3) of the internal elastic membrane, which is a homogeneous elastic and fenestrated layer, wavy in outline, and separating the intima sharply from the media. The *media* consists of circular, smooth muscle-fibers in regular arrangement, and of elastic fibers. The vasa vasorum pass through it. The *adventitia* consists of fibrillar connective tissue and elastic fibers. The *vasa vasorum* are minute arteries piercing the arterial wall from without for its nourishment. These arteries pierce the adventitia and media, but only their capillaries pass through the fenestra of the elastic membrane, and these stop short just beneath the epithelium.

In the **large arteries** the layers are less sharply defined. The intima is thicker and less distinct, the elastic tissue, arranged in irregular lamellæ, forms a predominant part of the media, and the adventitia also has an increased amount of elastic tissue.

## CHRONIC ARTERITIS.

Chronic arteritis (*atheroma*, *endarteritis*) is a chronic productive inflammation of the arteries throughout the body, associated with a degeneration of their walls. Certain conclusions arrived at by Thoma are generally accepted:

1. Every long-continued slowing of the blood current causes contraction of the middle coat of the aorta, and when this is not sufficient to accelerate the blood current it leads to a growth of connective tissue in the intima.

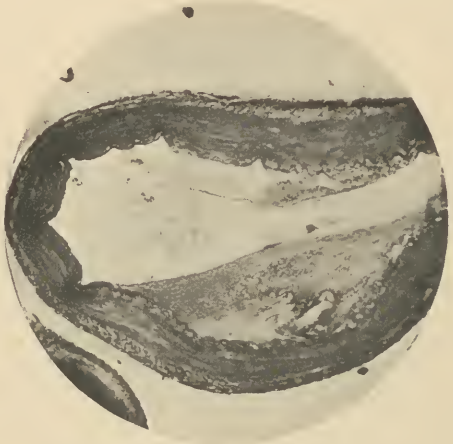

FIG. 95.—OBLITERATING ENDARTERITIS.
New connective tissue, rich in cells, has formed on the opposite sides of the artery *within* the elastic membrane.

2. Primary diffuse and nodular arteriosclerosis depends upon a weakening of the wall of the blood-vessel, due to constitutional conditions. This is followed by dilatation of the vessel, slowing of the blood-stream, and then to the growth of connective tissue in the intima.

3. Secondary arteriosclerosis is caused by slowing of the blood current, produced by changes of the circulation in the capillary vessels.

It will be necessary to describe arteriosclerosis as it occurs in the larger vessels, then as it occurs in the smaller vessels, because of characteristic differences.

### The Aorta.

Irregularly scattered over the surface of the aorta, and especially about the orifices of its branches, are numerous flat projections, round or oval in form, translucent or yellow in color, and seldom over ½ of an inch in diameter. The nodules may remain firm or break down and undergo fatty or calcareous degeneration. The change is beneath the endothelium, and consists in a degenerative infiltration of the media and adventitia around the vasa vasorum. A small round-cell proliferation is set up in the subendothelial tissues, with thickening and the formation of a button. This

Fig. 96.—Obliterating Endarteritis.

A very large growth of new tissue has taken place beneath the elastic membrane and around the entire circumference. The dark portion in the center is a blood-clot.

button seems to form by way of compensation to fill up the depression made by the degeneration in the media, thereby presenting a smooth inner surface during life. When such a nodule breaks down it forms an *atheromatous abscess*, and this in turn may break through the intima and form an *atheromatous ulcer*.

### The Smaller Arteries.

In the smaller arteries the changes are somewhat different. There may be simply a greatly increased growth of endothelial cells; or, as in syphilis and Bright's disease, there may be an obliterating endarteritis where there is a growth of connective tissue

from the endothelium. This new tissue is, at first, rich in large branching and round-cells, but later it becomes more fibrous. The growth of new tissue is usually greater on one side of the lumen than on the other, and as a rule there is a gradual obliteration of the lumen of the artery. In other cases the proliferation of cells, and especially of connective tissue, takes place immediately beneath the endothelium, and renders the inner coat so thick that there is great narrowing, but not an entire obliteration, of the lumen,

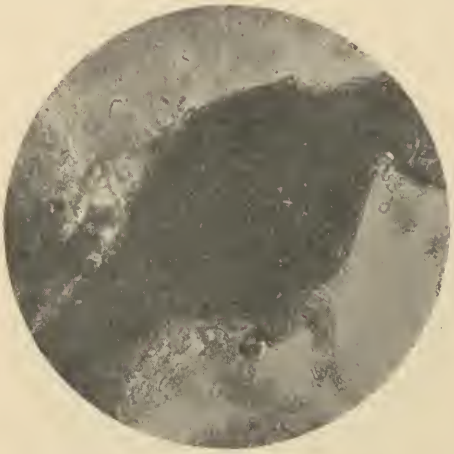

FIG. 97.—MILIARY ANEURYSM OF THE BRAIN OCCURRING IN SYPHILIS.

because the endothelium remains in place. Added to this there is usually an associated thickening of the muscular and outer coat with atrophy of the muscle-cells, or these outer coats may become fibrous and sclerotic.

In old age the arteries may undergo very extensive calcareous degeneration following destruction of the media, and be converted into rigid tubes (pipe-stem arteries). In such cases there are many atheromatous abscesses, and these break through to form ulcers.

## THE KIDNEY.

### Normal Structure.

The kidney in longitudinal section is seen to consist of three zones: (1) an outer cortical zone, which forms a comparatively thin shell over the entire surface of the organ; (2) a medullary or pyramidal portion, which appears as seven to ten red inverted cones with their apices near together in the pelvis, and (3) the pelvis of the kidney, which is a funnel-shaped cavity for the reception of the secretion of the organ. The kidney is surrounded by a firm, dense

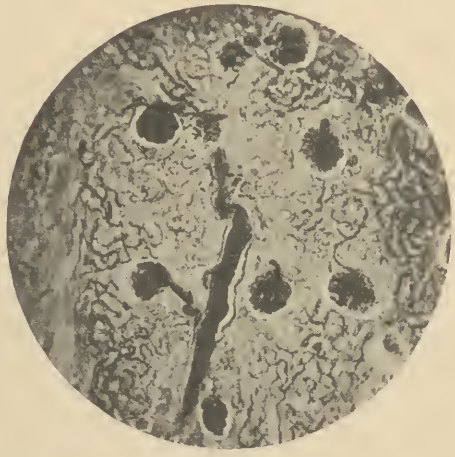

Fig. 98.—An Injected Interlobular Artery with the Glomeruli Supplied by it.

capsule which may be stripped from the surface, leaving it smooth and slightly mottled. The apex of each pyramid terminates in the pelvis, presents many minute openings, and is called the **papilla**, while from the base of the pyramid many narrow, tapering rays project out into the cortex to form the **medullary rays.** The cortical portion not only forms an outer layer between the base of the pyramid and capsule, but extends down between the pyramids, even to the papillæ, forming the so-called **interpyramidal cortex.**

The **uriniferous tubules** constitute the parenchyma of the organ, and are very complicated in their course. Each tubule is a simple tubular gland with a basement membrane and a single row of epithelial cells. At the exit from the glomerulus the tubule is nar-

rowed to form the **neck**, but it at once widens out to form the **convoluted tubule.** This winds around in the cortex and finally approaches a medullary ray, where it becomes very narrow and descends in a straight course for a variable distance into the pyramidal portion as the **descending arm of the loop of Henle.** It then turns sharply upon itself, broadens out, and returns to the medullary ray as the **ascending arm** of the loop of Henle. From this point the tubule again becomes convoluted and broad as the **intercalated** or **second convoluted tubule,** which is also in the cortex, then it returns to the medullary ray once more, enters the narrow **straight uriniferous tubule,** turns directly downward

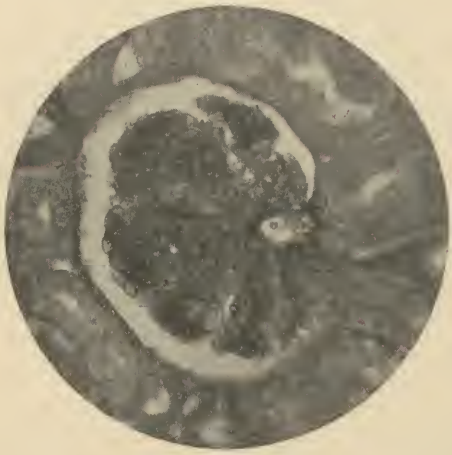

FIG. 99.—NORMAL KIDNEY GLOMERULUS, SHOWING DISTINCT LOBULATION OF THE TUFT.

through the pyramid, joins other straight tubules dichotomously, and opens in the apex of the papilla.

It will be apparent that each medullary ray made up of straight tubules and the upper portion of the loop of Henle will form a center about which all of the essential structures of the kidney are arranged. Such an area of kidney substance forms in this way an indistinct **lobule** with straight tubules in the center, convoluted tubules as the middle zone, and the Malpighian bodies in the periphery supplied by the *interlobular* arteries.

The **epithelial cells** of the *convoluted* and *intercalated* tubules are similar, and consist of large, granular, striated cells, ill defined in outline, and almost filling the lumen. The narrow *descending arm*

of the loop of Henle is lined by flat, transparent cells whose nuclei project into the lumen of the tubule, while those of the *ascending arm* are cuboidal and granular. The cells of the *straight collecting tubules* are cuboidal, transparent, and contain large nuclei, but near the papilla they become cylindrical.

The **glomerulus,** or **Malpighian body,** is really a spherical dilatation at the end of the tubule. It is surrounded by a capsule (*Bowman's capsule*), which is continuous with the membrana propria of the tubule, and is lined by a single layer of a very thin, transparent epithelium, which is also continuous with the tubular epithelium. Upon the side of the glomerulus, opposite the attachment of the tubule, a small artery (the *vas afferens*) passes through the capsule and divides into innumerable capillary loops, which are intertwined to form a capillary tuft, almost filling the cavity of the glomerulus. The blood is returned by another artery (*vas efferens*), which passes out alongside the entering one. This capillary tuft is covered by a layer of epithelial cells, similar to and continuous with that lining the capsule, and it will be evident that the glomerulus could be compared to an inverted tip of a glove finger.

**The Blood-vessels.**—The renal artery breaks up at once into small branches, which run up along the sides of the pyramids, enter the substance of the kidney at their base, and form a series of arches along the boundary line between the cortical and the medullary or pyramidal portions. From the convex side of the arch the *interlobular arteries* run up into the cortex between the medullary rays and give off frequent short lateral branches, which enter the glomeruli as the *vas afferens.* These arteries enter the glomerulus, break up into the capillary tuft, and the blood is again collected in the *vas efferens*, which still carries arterial blood; this artery again breaks up into a capillary network, which surrounds the convoluted tubules of the immediate area, the medullary ray, and a part of the base of the pyramid; then the blood is collected into interlobular veins. The medullary portion of the kidney is supplied by the capillaries just described, by the arch itself, and by the *vasa recta*, which descend from the concave side of the arch and spread out into a fine, long-meshed capillary network.

The connective tissue of the organ is, for the most part, not demonstrable.

## ACUTE CONGESTION OF THE KIDNEYS.

Acute congestion of the kidneys occurs after surgical operations, certain poisons, extirpation of one kidney, and overexertion. It is not a fatal condition, hence we must study experimental kidneys. These show an engorgement of the veins and capillaries, the tubule cells are flattened, and there is an exudation into the stroma and tubules of serum and red blood-cells.

## ACUTE DEGENERATION OF THE KIDNEY.

(*Acute Bright's Disease, Parenchymatous Degeneration.*)

Acute degeneration of the kidneys is the lesion of the kidney found in the acute infectious diseases, and in poisoning by phos-

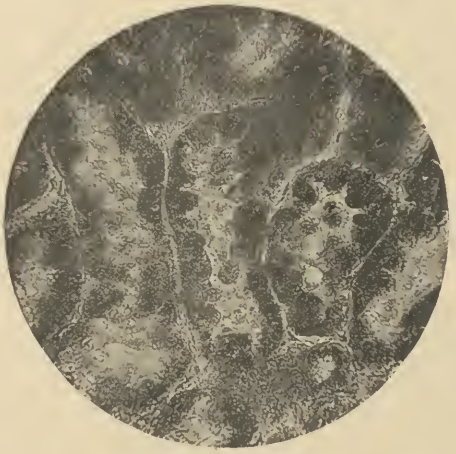

Fig. 100.—Acute Degeneration of the Kidney.

phorus, mercury, and arsenic. The changes produced are confined to the convoluted tubules, and vary with the quantity and virulence of the poison. (1) With a small dose the cells are merely swollen and very granular, and may return to the normal. There may be congestion of the vessels and some exudation of serum. (2) Larger doses will cause an infiltration of the cells, with albuminoid matter and fat, and there will often be a breaking down and disintegration of the cells. (3) Very large doses cause a coagulation-

necrosis, or a complete disintegration of the cells, and with this change there is intense congestion, an exudation of serum, the formation of casts, and a growth of new cells to replace those that are broken down. The kidneys are enlarged, the cortical portion is thick and pale, the organ is congested.

## ACUTE EXUDATIVE NEPHRITIS.

(*Acute Bright's Disease, Parenchymatous Nephritis, Tubal Nephritis, Catarrhal Nephritis.*)

Acute exudative nephritis occurs after exposure to cold, without apparent cause, or complicating the infectious diseases, especially scarlet fever.

*Macroscopically:* In the mild cases the kidneys look normal, but

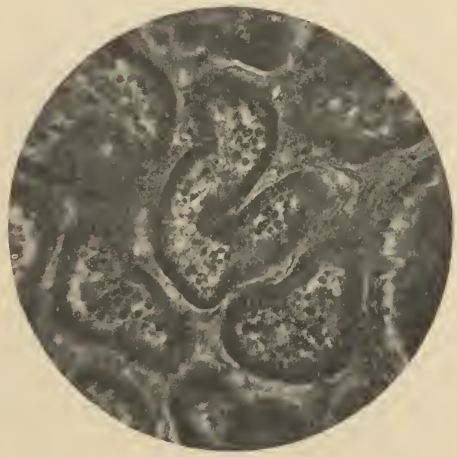

FIG. 101.—ACUTE EXUDATIVE NEPHRITIS.

The tubules are filled by fibrin and pus-cells; the stroma is edematous and contains pus-cells.

in the severe cases the kidney is large and smooth, the cortex is thick and white or greatly congested, the stroma is infiltrated, and the kidney is succulent and wet.

*Microscopically,* the **capsule** is not thickened; the **interstitial connective tissue** is unchanged except for infiltration with serum and white blood-cells. The **tubules** are empty or contain the coagulate exudate represented by cylinders of hyaline material and white blood-cells. There may be a dilatation of all the cortex

tubes. The epithelium is flattened or swollen and granular, or it is degenerated and detached from the walls. The capsular cells of the **glomerulus** are greatly swollen, especially at the point of tube entrance. The cavity of the glomerulus contains coagulated material, red and white blood-cells. In the capillary tufts the cells covering the tuft as well as the endothelial cells of the capillaries increase in number and become much swollen. In this way the tuft is increased in size, the outline of the individual capillaries is lost, but the main division of the tuft may still be seen.

In certain severe cases there is an excessive exudation of white blood-cells into the tubules, stroma, and glomeruli, which are not

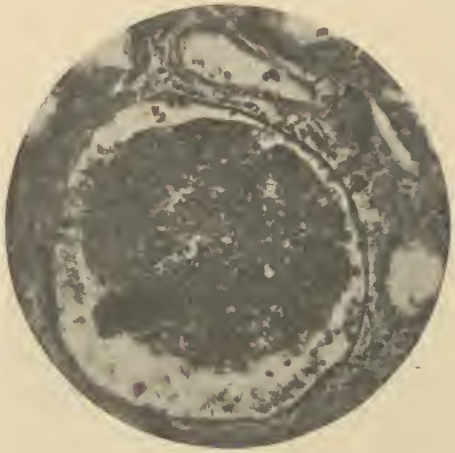

FIG. 102.—GLOMERULUS OF ACUTE EXUDATIVE NEPHRITIS.
The tuft shows great increase in the cells, an obliteration of all lobulation, and the space around it is filled by pus-cells.

equally distributed throughout the kidney but are collected in small foci having the appearance of small abscesses.

The lesions of an acute exudative nephritis are all transitory and the kidney may return to normal.

## ACUTE PRODUCTIVE NEPHRITIS.

(*Acute Bright's Disease*, *Parenchymatous Nephritis*, *Glomerulonephritis*, *Scarlatinal Nephritis.*)

Acute productive, or diffuse, nephritis follows scarlatina, diphtheria, pregnancy, exposure to cold, etc. *Macroscopically*, the

kidney is increased in size, the capsule is not adherent, the surface is smooth, the cortex is thickened and red or white in appearance.

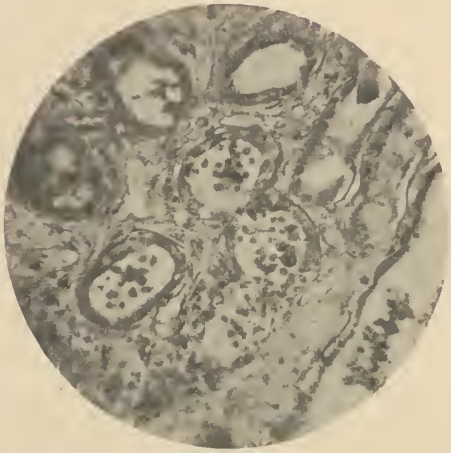

FIG. 103.—ACUTE PRODUCTIVE NEPHRITIS FOLLOWING SCARLET FEVER.
The tubules are filled by pus-cells, but between the tubules there is a large growth of new connective tissue.

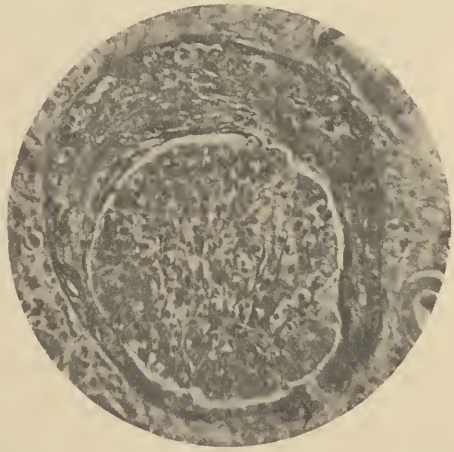

FIG. 104.—GLOMERULUS OF ACUTE PRODUCTIVE NEPHRITIS.
There is an increase in the number of cells covering the tuft and an extreme growth of capsular cells.

*Microscopically*, the **capsule** is somewhat thickened. The **interstitial connective tissue** or **stroma** shows either diffuse thickening, patches of new tissue, or long wedges running up into the

cortex along the arteries. The **tubules** show the changes of an acute exudative nephritis. The capsule of the **glomerulus** is thickened, the *capsule cells* are swollen and so much increased in number as to press on the tuft, and there is also an increase in the number and size of the cells covering the tuft. This change is confined to those glomeruli which are supplied by the thickened artery found in the wedge of new connective tissue.

The connective-tissue deposit and the glomerular change is permanent and progressive, and the condition is apt to be followed by a chronic nephritis. The connective tissue is present from the start, and its formation is not preceded by an acute exudative nephritis. A

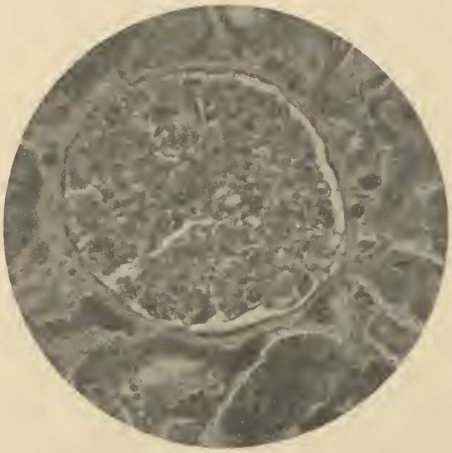

FIG. 105.—CHRONIC CONGESTION OF THE KIDNEY.

good clinical working rule to follow in differentiating this form of nephritis from the acute exudative is found in the fact that postscarlatinal nephritis and any *subacute* primary nephritis in a person over twelve years old is nearly always of the acute productive type; that nephritis following diphtheria and pregnancy is often of this type, while *all other* cases of acute nephritis are of the acute exudative type.

## CHRONIC CONGESTION OF THE KIDNEYS.

Chronic congestion of the kidneys is dependent upon obstructed circulation from heart lesions, aneurysm, emphysema, and fluid in the chest cavity. *Macroscopically*, such kidneys are large, heavy,

and very hard, dark red in color, the capsule is not adherent, and the surface is smooth. *Microscopically*, the **capsule** is sometimes a little thickened. The **stroma** is normal; the **tubules** are unchanged or the cells may be flattened. The diagnostic feature is the **glomerulus**, and in this the capsule cells are unchanged; but the capillaries of the tuft are *dilated*, their walls are thickened, and the cells covering them are swollen. The **arteries** of the kidney, as a rule, are unchanged. Chronic nephritis is very apt to follow chronic congestion.

## CHRONIC DEGENERATION OF THE KIDNEYS.

(*Chronic Bright's Disease, Chronic Parenchymatous Nephritis, Fatty Kidney.*)

Chronic degeneration of the kidney occurs in obstructed circulation, in old and feeble persons, and in chronic diseases, especially cancer and phthisis. *Macroscopically*, the kidneys are often very large, the capsule non-adherent, the surface smooth, the cortex thick and white, the pyramids red. *Microscopically*, the **capsule** is not thickened, there is no change in the **stroma**, the **tubular** epithelium is swollen, granular, or infiltrated with fat. The **glomeruli** show dilated and thickened capillaries. There is no change in the **arteries.**

## CHRONIC NEPHRITIS.

Various authors have given names to the varieties of chronic nephritis which are dependent entirely upon some appearance of the kidney, or upon the predominance of some one change in the microscopical structure of the organ. This has led to much confusion, and made it impossible to judge, from the clinical history of a patient, just what lesion really exists in the kidney. Furthermore, every physician who has made many autopsies must have observed that with a given clinical history the autopsy findings vary greatly. A large white kidney may be expected, yet on autopsy it will be found normal in size, or contracted, and vice versa.

Hence it is taught by Dr. Delafield that in **all** forms of chronic nephritis there is a growth of new connective tissue in the stroma,

destruction of tubular epithelium, and a permanent change in the glomeruli. Also, that the predominance of any one element in these changes, or the degree of change present, bears no correspondence to the clinical symptoms given during life. However, the presence or absence of exudation from the blood-vessels does bear a very distinct relationship to the clinical picture. Consequently, a division is made into (1) a chronic productive nephritis **with** exudation, where the symptoms are: urine diminished in amount, low in specific gravity, and full of albumin and casts; there is decided anemia, and a waxy skin; edema of the extremities regularly, dyspnea, no temperature, headache, sleeplessness, acute or chronic uremia, and hypertrophy of the left ventricle. (2) Chronic productive nephritis **without** exudation, where the urine is increased in amount, 1010, contains no albumin (except occasionally) and no casts; edema of the extremities is absent, the skin is not anemic and waxy, unless it be very late; temperature 102° to 108° F. in attacks; dyspnea is very frequent from contracted arteries; a catarrhal gastritis is present; acute uremic attacks are frequent; the left ventricle is regularly hypertrophied, and the arteries contracted.

## CHRONIC PRODUCTIVE NEPHRITIS WITH EXUDATION.

(*Chronic Bright's Disease, Parenchymatous Nephritis, Chronic Glomerulonephritis, Large White Kidney, Chronic Diffuse Nephritis.*)

Chronic productive nephritis with exudation follows acute exudative nephritis, chronic congestion, and chronic degeneration of the kidney, and is found in the prolonged and wasting diseases. The kidneys vary much in size and appearance. They may be very large, with white or mottled cortex; normal in size with these appearances; small, with a thin cortex and large red pyramids; normal in every appearance, or very small and contracted. The capsule is adherent or not, and the pyramids are usually red. *Microscopically*, the **capsule** is much thickened; wedges of new connective tissue run into the cortex from the capsule, or it forms along thickened arteries and around each tubule. The amount of this new connective tissue increases as time goes on. The **tubules** are normal, atrophied or dilated, and contain casts; their epithelium

is swollen and granular, fatty, and broken down or flattened. The capsule of the glomerulus shows connective-tissue thickening; the

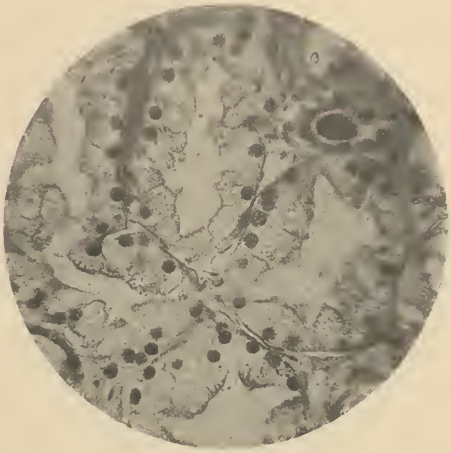

FIG. 106.—LARGE WHITE KIDNEY.

Photograph from that portion of the section showing the change in the tubule cells without an increase in the stroma.

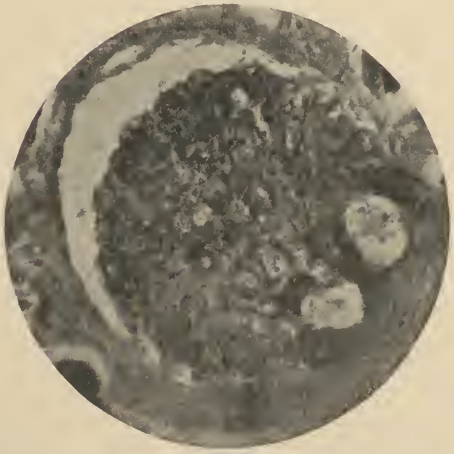

FIG. 107.—GLOMERULUS OF CHRONIC PRODUCTIVE NEPHRITIS.

capsule cells multiply greatly and press on the tufts. The cells covering and lining the capillaries may also increase in size and number to help in the obliteration of the tuft to a fibrous ball. The

walls of the capillaries may be dilated and thickened when the nephritis follows chronic congestion. The **arteries** are usually the seat of an obliterating endarteritis.

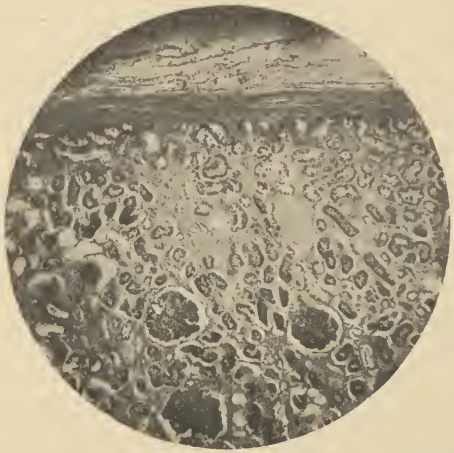

FIG. 108.—CHRONIC PRODUCTIVE NEPHRITIS WITH EXUDATION.
The thickened capsule is seen above, and a growth of new tissue between the tubules.

## CHRONIC PRODUCTIVE NEPHRITIS WITHOUT EXUDATION.

(*Chronic Bright's Disease, Cirrhosis of the Kidney, Granular Kidney, Interstitial Nephritis.*)

Chronic productive nephritis without exudation usually occurs after forty-five years of age, and seems to be caused by syphilis, gout, chronic alcoholism, lead poisoning, and is often associated with emphysema, endarteritis, and cirrhosis of the liver. *Macroscopically*, the kidney is usually very small, the capsule is adherent, and removes portions of kidney substance with it, the surface rough and nodular and may show cysts, the cortex thin and red or gray, consistency tough, dense, and fibrous. *Microscopically*, the **capsule** is thickened, the connective-tissue growth in the **stroma** is very extensive, especially in the cortex, and has the arrangement of wedges, or is diffuse and increases in amount as the disease goes on. The **tubules** are obliterated and atrophied, or between areas of connective tissue they may be dilated even to a cystic condition. The epithelial cells are flattened, cuboidal, or swollen and degenerated. The changes in the **glomeruli** are very

similar to those of the exudative form, and they are found in all stages of atrophy. This atrophy is dependent upon: (1) The con-

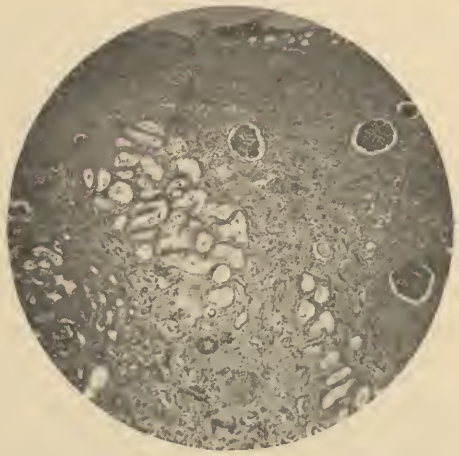

FIG. 109.—CHRONIC PRODUCTIVE NEPHRITIS WITHOUT EXUDATION.
The capsule above is extremely thickened. Dense wedges of tissue run into the kidney substance, leading to every grade of atrophy in the glomeruli and tubules.

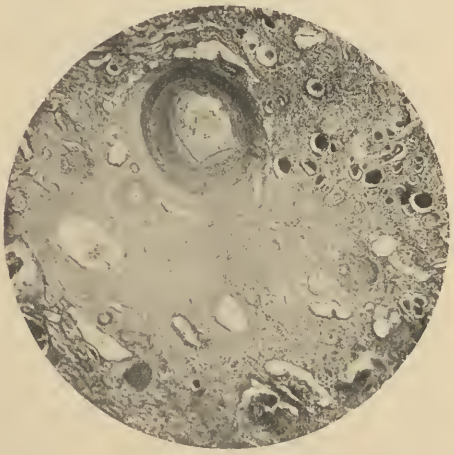

FIG. 110.—EXTREME ATROPHY OF THE KIDNEY.
Showing advanced arteriosclerosis of glomerular change.

nective-tissue thickening about them; (2) the growth of intra-capillary and of capsule cells; (3) the obliteration of the small arteries supplying them; (4) amyloid degeneration taking place in

the walls of the capillaries. The arteries of the kidneys are the seat of an obliterating endarteritis.

Associated with the changes in the kidney there is *regularly* present an arteriosclerosis, and a hypertrophy of the left ventricle of the heart.

## SUPPURATIVE NEPHRITIS.

Surgical kidney results most frequently from a transmission of infection through the ureter and is secondary to enlargement of the prostate, stricture of the urethra, renal calculus, and cystitis. From

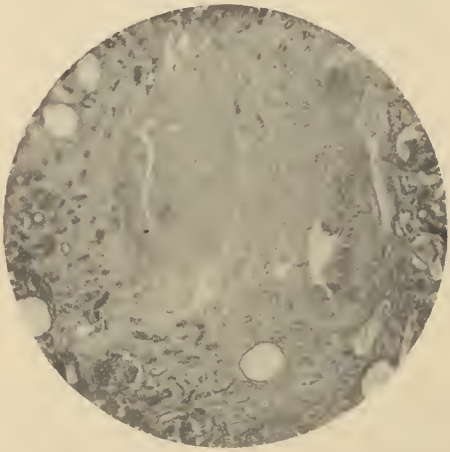

FIG. 111.—PYEMIC ABSCESSES OF THE KIDNEY.

the mucous membrane of the pelvis the bacteria are carried up into the cortex, where they form circumscribed abscesses.

The kidney is swollen, soft, friable, and presents, on its surface, yellow elevations surrounded by an area of hyperemia. The abscesses appear as round, pyramidal, or elongated collections of pus, few in number, or the kidney is riddled by confluent abscesses, or large abscesses communicate with the pelvis to form pyonephritis. *Microscopically*, the process is seen to begin by a collection of small round-cells along the interlobular arteries at the base of the pyramids and between the tubules. From this point the process is one of disintegration of the kidney elements and the formation of abscess cavities which contain pus, bacteria, and broken-

down cells, and their wall consists of an inner necrotic area, an area of degeneration and infiltration by pus-cells, and a surrounding area of hyperemia.

The infection of the kidney by the pus-forming bacteria may also be embolic or metastatic in pyemia and malignant endocarditis. In this case the septic emboli lodge and produce multiple abscesses similar in every way to those just described.

## TUBERCULAR NEPHRITIS.

Tubercular disease of the kidney usually begins in the mucous membrane of the pelvis where miliary tubercles and granulation tissue are formed. From this point it extends first to the pyramids, then to the cortex, and invades a large part of the organ, causing decided enlargement. The new tissue caseates very early, so that the whole organ soon consists of a number of cysts full of caseous material. Suppuration may be added to the tubercular inflammation, or the kidney may previously have been the seat of a chronic nephritis. One kidney is affected, as a rule.

## CYSTIC KIDNEY.

In the *congenital* cystic kidney either one or both organs are entirely converted into one large mass of cysts lined by a single row of cells and separated by fibrous septa and compressed kidney substance. These seem to be formed from dilated tubes and glomeruli. In the *adult*, cystic kidneys present a single cyst, or in the atrophic form of chronic diffuse nephritis there may be great numbers of small cysts caused by dilatation of the obstructed tubules. There is also a form occurring in the adult where the organs are converted into a mass of cysts and give no symptoms until nephritis sets in.

## HYDRONEPHROSIS.

Dilatation of pelvis and calices of the kidney by collected urine occurs congenitally, without apparent obstruction, or it is the result of an obstruction of the ureter from tubercular disease, calculi, or

pressure from without. The kidney is lobed, the cortex is thin, and either unchanged or the seat of a chronic nephritis. Suppuration may supervene.

### WAXY KIDNEY.

Amyloid degeneration affects the glomeruli and the walls of all the blood-vessels of the organs.

FIG. 112.—AMYLOID DEGENERATION OF THE KIDNEY.
The glomeruli and arteries in the field show extensive amyloid change.

### TUMORS.

The most common *tumors* of the kidney are the sarcoma, the papillary and alveolar adenomata. Myoma, lipoma, fibroma, carcinoma, and myxoma are occasionally found.

## THE ORGANS OF GENERATION.

### FEMALE GENERATIVE ORGANS.

#### ENDOMETRITIS.

In acute catarrhal endometritis the mucous membrane is swollen and hyperemic. There is a small, round-cell infiltration of the mucosa and a dilatation of the vessels. The glands are dilated and

the epithelium is in a catarrhal condition, swollen and granular, or it is desquamated, or the surface may be covered by a mucopurulent exudate.

FIG. 113.—CHRONIC ENDOMETRITIS.

There is observed a tortuosity of the glands, an increase in their number, and a growth in the interglandular tissue.

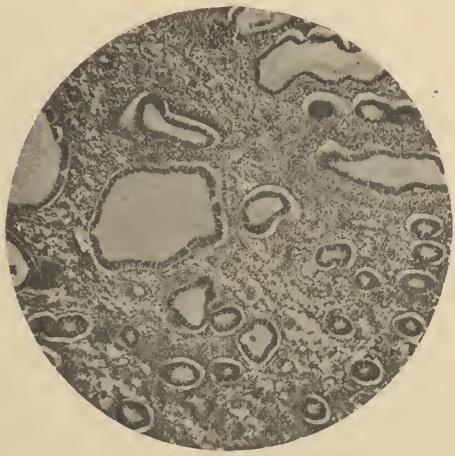

FIG. 114.—ADENOMATOUS HYPERPLASIA OF THE UTERINE MUCOSA.

Newly-formed ducts of every size and shape are found, each lined by a single row of epithelial cells. The interglandular tissue is increased.

## CHRONIC ENDOMETRITIS.

In chronic endometritis there may be a continuation of the process found in the acute type; or there may be an **hypertrophy**

of the mucosa with an increase in the number of glands, a branching of the old, a fibrillation and marked increase in the cells of the interglandular connective tissue, and a formation of papillary *fungous* or *polypoid outgrowths*. The blood- and lymph-vessels are increased in number and size. The cells lining the glands are always arranged in a single row, do not break through the basement membrane into the intertubular connective tissue, and do not invade the muscular coat. When an increase in connective tissue accompanies this hypertrophy forming distinct elevations or fungosities, it is sometimes called **adenomatous hyperplasia** or **benign adenoma.** It is now universally agreed, however, that the only adenomata of the uterine mucosa are adenocarcinoma, because of their great tendency to assume, even at an early stage, a cancerous nature.

There is also an **atrophic form of chronic endometritis** where the glands and their epithelium are diminished in size, the interstitial lymphoid tissue has fewer cells and becomes densely fibrous, even to complete obliteration of the glands. This is also the condition left in the mucosa after the application of caustics.

### Septic Endometritis.

After delivery, the inner surface of the uterus is denuded of epithelium, rough, and covered with necrotic tissue, blood-clots, and serum, and, furthermore, the veins and lymph sinuses are open. Germs gaining entrance into the uterine cavity at this time find, in these clots, necrotic tissues, etc., kept as they are at body temperature, the very best culture medium. Septic germs thus cultivated produce a very severe inflammation, with rapid necrosis of the mucous membrane. Abscesses are formed along the course of the veins and lymphatics, the muscularis is softened and swollen, and germs are found throughout its thickness. Pelvic abscess may form in the broad ligament, or other structures, or a pelvic or general peritonitis may be set up. These same conditions may arise also from the introduction into the uterine cavity of infected instruments, or from any foul manipulation by which germs may be introduced or the mucosa injured.

In the *chronic form* of septic endometritis necrosis is absent, but all other symptoms of the acute form are present in a lesser degree. Pelvic peritonitis, cellulitis, salpingitis, and ovaritis are constant accompaniments.

### Gonorrheal Endometritis.

Gonorrheal endometritis presents the same condition as the septic form, but there is no necrosis, the gonococci do not penetrate, as a rule, deeper than the epithelium, and the systemic infection is less severe. Much pus is formed, and the important complication is salpingitis.

### Erosion or Ulceration of the Cervix.

This is a condition following laceration of the cervix, or a cervical catarrh, where there appears, on the vaginal portion of the cervix, a more or less extensive beef-colored area, which is papillary or velvety in appearance, and bleeds very easily. The surface is thrown into papillary folds, which are covered by a single row of cuboidal epithelial cells continuous and identical in character with those covering the cervical canal. At the border of limitation from flat vaginal cells the transition seems very gradual from one type of cell to the other. It seems to be an extension of the cuboidal cervical epithelium out over cervical tissue which has been denuded of its squamous epithelium. Deeper down in the tissues are irregular alveoli, lined by the same kind of epithelium, and these would seem to be the deep infoldings between the papillæ. Associated with this there is a proliferation of the connective tissue just beneath the surface. The explanation of the tendency to form erosions after laceration of the cervix is to be found in the more rapid proliferation of the cuboidal than of the squamous epithelium, and a consequent growth over the denuded surface of the former type of cell.

### Tumors of the Uterus.

Pure **fibromata** of the uterus are comparatively rare, and those tumors usually spoken of as "fibroids" are either pure myomata, or fibromyomata.

The **myomata** are the most common of all uterine tumors, and vary in size from that of a marble, to a nodulated tumor occupying the entire abdomen. Their seat of growth is either subperitoneal, intraparietal, or submucous.

To the **adenomata** belongs that type of growth from the mucous membrane which lies on the border-line between hypertrophic endometritis and carcinoma. The transition is so gradual, and many of these adenomatous growths are so distinctly malignant in

every sense, that many observers avoid the use of the name adenoma entirely, and regard all such tumors as adenocarcinomata, or plainly as carcinomata. If the name be retained, however, it must be remembered that certain forms are very malignant in character, while others are very prone to change to a malignant character. They develop from a simple hyperplasia of the mucous membrane, and where there is a new growth of glandular tissue, with the production of numbers of new glands. These glands are branching, irregular, may have a dilated follicular structure, and regularly have a well-defined lumen, with the cells arranged in a single layer on a membrana propria. They form projections or

FIG. 115.—CARCINOMA OF THE CORPOREAL ENDOMETRIUM.
The section is one mass of epithelial cells in atypical arrangement.

papillary new growth on the surface of the mucous membrane, and invade the submucosa.

The most common form of **carcinoma** is epithelioma.

**Epithelioma** occurs in the cervix, and is either flat and ulcerating, a fungous or cauliflower growth, or in the form of nodules in the cervix, dependent upon its origin, which may be in the epithelium of the rete Malpighii, the cylindrical epithelium within the cervix, or the glandular epithelial cells. Such growths extend rapidly or slowly to the vagina, hypogastric glands, rectum, and pelvic bones.

**Carcinoma** of the **body** of the uterus originates in the glands of the corporeal endometrium. The epithelial cells increase in

number to form more than a single row, or to complete filling of the lumen of the gland duct, they break through the membrana propria into the interglandular tissue to form nests and columns, and invade the muscularis. The close relationship mentioned above between hypertrophic endometritis, adenoma, and carcinoma need not be repeated.

## *THE OVARY.*

### Acute Oöphoritis.

In acute oöphoritis occurring with or independent of the puerperal condition, there is a swelling and congestion of both ovaries, the stroma is infiltrated with serum and pus, and the follicles are enlarged or become cystic. Abscesses often form in one ovary, and the organ is usually bound down by bands resulting from the accompanying peritonitis.

Fig. 116.—Chronic Oöphoritis.

The structure is entirely of dense connective tissue and blood-vessels. The clear space above is a portion of a large cyst.

### Chronic Oöphoritis.

Chronic oöphoritis usually follows an acute inflammation, or long-continued obstruction of the circulation. The organ is enlarged and loose in texture, or dense, new connective tissue is formed throughout the organ, or in the capsule alone. The

vessels are dilated, and cysts may be formed to such an extent as to make up the main mass of an enlarged ovary. The organ may be atrophied, and made up of a mass of fibrous tissue and thickened arteries. A hyperplasia of the cells in the corpus luteum often gives rise to a convoluted nodular mass in the ovary that may seem like a tumor. Ovaries, the seat of a chronic inflammation, are usually bound down by adhesions.

### Tumors of the Ovary.

Fibroma, sarcoma, chondroma, and carcinoma are among the tumors occurring in the ovary, but they are not common.

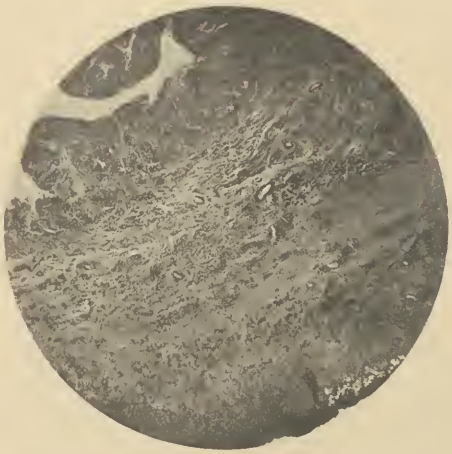

Fig. 117.—Pyosalpingitis.

The mucous membrane is shown above thrown into polypoid folds, while the entire thickness of the tube to the peritoneal surface below is dense fibrous tissue infiltrated by small round-cells.

**Adenomata** are the most common and important tumors. They occur as *multilocular cysts* or *glandular cyst adenomata*, where the multiple cyst walls are smooth and lined by a single row of epithelial cells, or as *papillary cyst adenomata*, where there are papillary outgrowths and tufts from the wall of the cyst.

*Follicular cysts* arise from dilatation of Graafian follicles. *Dermoid* cysts are frequent.

### Salpingitis.

**Catarrhal Salpingitis.**—There may be simple catarrhal inflammation of the Fallopian tubes, associated with endometritis. The

lesion itself is insignificant, but it most often results in chronic thickening, with adhesions and obliteration of the lumen in certain parts, with subsequent dilatation from collected secretion to form hydrosalpinx, or, if infection gains entrance, pyosalpinx.

**Pyosalpingitis.**—Pyosalpingitis usually results from gonorrheal infection through the uterus. The fimbriated extremity at once becomes closed, and is glued to any structure with which it happens to lie in contact. The walls of the tubes either undergo great connective-tissue thickening, with some destruction of the mucous membrane and little or no collection of pus in their lumen, or with closure of the uterine end of the tube, as well as the fimbriated end, they are dilated to almost any degree by pus. In both varieties adhesions form to a varying degree, but in the latter all the pelvic organs are firmly bound down, and the abscess may also involve the adjacent ovary, the broad ligament, intestine, or bladder.

## THE BLOOD.

### Normal Structure.

The normal human blood has for its structure a definite number and proportion of red blood-corpuscles, white blood-corpuscles, and blood plates suspended in a blood-plasma which, in turn, contains the elements necessary to form fibrin and serum.

The *red blood-cells* are round, biconcave discs, with a thickened rim. They are seven mm. in diameter, number 5,000,000 to the c.mm., and contain the oxygen-carrying hemoglobin, which gives them the red color.

The *white blood-cells* are larger, lighter in color, spherical or elongated, and have one or more nuclei. The varieties are as follows:

(1) **Small lymphocytes,** which constitute from 20 to 30 per cent. of the white corpuscles of the blood, and represent the youngest type of cell. It has a large nucleus, about the size of a red blood-cell, surrounded by a narrow rim of protoplasm.

(2) The **large, mononuclear leukocyte** is a result of the growth of the former type, with an increase in the amount of protoplasm and the size of the nucleus. The protoplasm is transparent and

non-granular. This variety constitutes four to eight per cent. of the total number of leukocytes.

(3) **A transitional form** of cells is also large, pale in color, and non-granular, but its nucleus is indented, or horseshoe-shape. It is merely one stage in development, and there is no other reason than the shape of its nucleus for giving it a name.

(4) The next stage in development from the small lymphocyte is the **polymorphonuclear neutrophile,** which represents the fully-developed cell, and constitutes 70 per cent. of the white blood-corpuscles. The nucleus of these cells is *very irregular* in shape, stains deeply and irregularly, and the cell protoplasm is filled by very fine granules which are stained only by neutral stains, such as Ehrlich's.

(5) An overripe cell, called the **eosinophile,** is next described as a cell very similar to the neutrophile, except that the cell is somewhat smaller, paler in color, and the protoplasmic granules are larger, spherical, and take only acid stains, such as eosin, acid fuchsin, etc. The cell is more loosely constructed, the granules may collect on one side of the cell, and their ameboid motion is very active. This variety constitutes 0.25 to 4 per cent. of the white cells.

(6) Certain very large cells called **myelocytes** are normally abundant in the bone-marrow, but wander only very occasionally into the normal blood. They have a regular oval or spherical nucleus which occupies almost the entire cell, *stains equally* throughout, and is eccentrically placed, so that it is in close contact with the surface of the cell. The protoplasm is filled by neutrophile granules, and it will be apparent from the description that this cell differs in this particular alone from the large lymphocyte, and from the polymorphonuclear neutrophile alone in the shape of its nucleus. Their size varies from 10 to 21 $\mu$.

## LEUKOCYTOSIS.

Leukocytosis is an absolute but temporary increase in the number of leukocytes in the peripheral blood over the number normally present. The normal number of leukocytes present in the blood is usually placed at 7500 per c.mm., or almost one to every 250 red cells, but it certainly may vary from 3000 to 10,500 in health with-

out assignable cause. Leukocytosis may be of two kinds: (*a*) where the relative proportions of the different varieties of cells remains unchanged, and (*b*) where the increase is entirely or mainly in the polymorphonuclear or adult leukocytes. The first class will include a great variety of conditions of physiological increase in the leukocytes, while the latter includes all pathological leukocytosis.

Thus, under the head of **physiological leukocytosis,** we find that in the new-born there is a high percentage of leukocytes; also that the number of these cells in the blood is so dependent upon food taken that it becomes of diagnostic value in testing the accuracy of statements that the patient eats nothing, or in determining the general nutrition, the rapidity of digestion, etc. It is important not to confuse such leukocytosis with pathological conditions. Other examples of physiological leukocytosis are found in the later months of pregnancy in primiparæ, after parturition, after violent exercise and cold baths.

The **pathological varieties,** as stated, differ in the fact that the increase is greater and takes place in the polymorphonuclear cells. Such a leukocytosis occurs after hemorrhage in many of the inflammatory conditions, in infectious diseases, in poisoning, and in malignant disease, and is caused by certain drugs. In inflammatory leukocytosis certain conclusions may be tabulated: (1) That the increase seems greatest in the purulent and gangrenous conditions than in the serous; (2) that the amount and duration of the leukocytosis has characteristic differences in different diseases; (3) that the amount of exudation taking place is not a measure of the leukocytosis present; (4) that there is no connection between fever and leukocytosis; (5) that acute spreading inflammations produce greatest increase.

## LYMPHOCYTOSIS.

Lymphocytosis is a relative increase in the lymphocytes or young cells of the blood, with or without an increase in the total number of leukocytes. Such an increase occurs in healthy infants, rickets, hereditary syphilis, scurvy, chlorosis, and pernicious anemia, anemia of syphilis, in splenic tumors, at the end of scarlet fever, delayed resolution of a pneumonia, and in lymphatic leukemia. Lymphocytosis, when combined with eosinophilia, is of

diagnostic value in obscure syphilis, and in the presence of swelling of the glands it is the main diagnostic sign of lymphatic leukemia.

## EOSINOPHILIA.

Eosinophilia is an increase in the eosinophiles of the blood. This increase is not peculiar to leukemia, but does occur in this disease. A definite connection, however, has been found between eosinophilia and diseases of the bones, the female genital organs, especially the ovaries, the skin, the sympathetic nervous system, the presence in the body of some one of the xanthin bases, and certain tumors of the spleen. Such an increase is of value in diagnosing puerperal mania as opposed to puerperal sepsis, a tumor of the genital organs from one not so connected, in diagnosing bone metastasis in malignant disease, syphilis (when the eosinophiles are combined with lymphocytes), obscure uric acid diathesis, and other diseases of the liver where a malignant tumor may be suspected. *Abundance* of eosinophiles renders the prognosis very good in chlorosis, pernicious anemia, after severe hemorrhage, scarlet fever, and scarlatinal nephritis.

## MYELOCYTOSIS.

Myelocytes in the blood are always pathological, inasmuch as they belong to the bone-marrow. They occur pathologically in leukemia, pernicious anemia, severe chlorosis, syphilis, malignant disease, uremia, etc.

## CHLOROSIS.

The changes in the blood of chlorosis are qualitative rather than structural. The red blood-cells may show a slight decrease in number, and the leukocytes somewhat of an increase, but the essential change is a lowered specific gravity of the blood and a very great decrease in the hemoglobin percentage contained in the red blood-cells. Thus, with the blood-corpuscles *normal* in number, there may be a hemoglobin percentage of 35. The red corpuscles may show a marked diminution in diameter, are pale in color,

sometimes show decided poikilocytosis, and nucleated cells may be present. Inasmuch as most of the changes possible in red blood-corpuscles may be found in the various grades of simple chlorosis, it will be well to describe these changes in full.

**Endoglobular changes** consist in the appearance within the red blood-corpuscle of variously shaped clear hyaline spaces. These change rapidly in the fresh state, but form sharply outlined spaces when dried.

**Poikilocytosis** in red blood-cells is a phenomenon where a lump appears in one side of a cell and becomes pointed; the cell takes on ameboid motions, and the battledore, club-shape, and horse-shoe-shape cells result. Some of these pointed projections may break off and move around actively. **Crenation** is the same process taking place outside the blood-vessels.

The normal red blood-cell can be stained only by acid stains, but in diseases the changes are such that the cell takes up two or three colors, either uniformly or in irregular manner. This is called a **polychromatophilic change.**

The red blood-cells may show changes in diameter, fail to form rouleaux, and may lose their hemoglobin entirely.

Important among the changes in the red blood-cells of anemia are the regenerative efforts which are expressed in the appearance of nucleated red blood-cells. These are of three kinds:

1. The **normoblasts** represent immature red blood-cells, which are abundant in the bone-marrow, and sometimes wander into the general circulation. The cell diameter corresponds to that of the ordinary red blood-cell, but a round nucleus is present which is one-half the diameter of the cell and stains deeply. The nucleus seems to be pushed out as the cell grows older.

2. **Megaloblasts** are cells that do not appear at any time or in any region of the normal human body. They are large cells appearing in the blood of certain anemias, but their nuclei are absorbed, and there is no tendency to regeneration as is expressed in the normoblast. Hence, it is degenerative, and is considered as a bad prognostic sign. It is 11 to 20 $\mu$ in diameter, and shows polychromatophilia; the nucleus is so large as to fill almost the entire cell, and is paler, and more evenly stained, than the normoblast. The protoplasm forms a colorless ring around the nucleus, and the entire cell is oval or globular.

3. **Microblasts** are much rarer than the preceding types, have

a nucleus similar to the normoblast, but the entire cell is very much smaller. Their prognostic significance is supposed to be that of megaloblasts.

Besides these typical forms of cells, there are a great number of variations which represent transitional or erratic forms, because no sharp lines can be drawn even between the three main varieties.

## PERNICIOUS ANEMIA.

The blood of pernicious anemia has a pale, watery appearance, is so fluid that it rapidly slips away when drawn with a needle, and it coagulates slowly. The red cells do not show rouleaux forma-

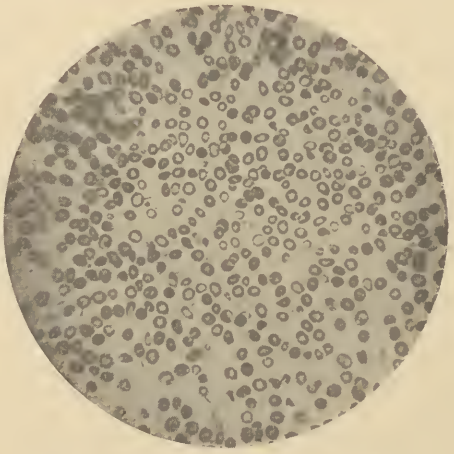

FIG. 118.—BLOOD OF PERNICIOUS ANEMIA.
Showing poikilocytosis and increased cell diameter.

tion; there is great variation in the size of the corpuscles; they are few in number, and often have a rapid, ameboid movement. The hemoglobin percentage remains relatively high, as a rule.

The *red cells* are diminished to a count of 1,200,000 at the time such a patient comes for treatment, but a lesser or very much greater reduction may be found at other stages of the disease. The diameter of the majority of cells is constantly increased, even to 20 $\mu$, and the macrocytes are much more numerous than the microcytes. The corpuscles are much deformed, the

characteristic shape being the horseshoe, battledore, and sausage shapes. They stain unequally, and have white streaks, transparent centers, or are polychromatophilic. The nucleated forms of the red blood-corpuscles are present in varying numbers, which can not be well estimated, but their character is more important than their number, inasmuch as two megaloblasts mean a graver prognosis than twenty normoblasts. The former mean a bad prognosis.

The *white corpuscles* show a decided diminution in number to 5000, as an average, and many reach 500 to the c.mm. A characteristic feature is a lymphocytosis (both small and large), the eosinophiles may be increased, and a few myelocytes are usually found.

The above characteristic points will most positively differentiate the disease from chlorosis. If the blood count is diminished to 1,000,000 in chlorosis, malignant disease, or other secondary anemias, there is always a leukocytosis by which we can rule out pernicious anemia. The abundance of myelocytes in leukemia as related to pernicious anemia serves as a diagnostic point when there is great resemblance in other points, especially in children, where there is a leukocytosis in all anemias.

## LEUKEMIA.

Leukemia is divided into the splenic-myelogenous form, showing a common splenic enlargement, and a lymphatic form, where the lymph-nodes are enlarged and the spleen but slightly. The blood changes differ essentially.

### Splenic-myelogenous Form.

The blood in this form is normal in color, flows sluggishly, coagulates slowly, and is spread with difficulty on a slide. The hemoglobin percentage is somewhat diminished. The *red blood-cells* are diminished to about 3,120,000, and the presence of the nucleated forms in large numbers is a very characteristic feature. The normoblasts predominate over the megaloblasts.

The *white blood-cells* show every grade of increase up to an equality in number with the red, but the average percentage is about one white to eight red. The cells are not ameboid because of the enormous predominance of **myelocytes**, which are not

amebic. This predominance forms an *absolute diagnostic feature*, and even though myelocytes are found in other conditions, the largest number ever found in such conditions is so much below the smallest number ever found in leukemia that there never can be the slightest question as to diagnosis. The polymorphonuclear cells show an absolute increase but a relative diminution, and show great variety of form. The *lymphocytes* are very markedly diminished in their relative proportion, and form from 30 to 70 per cent. of the white cells even though their absolute number is increased. They appear normal in structure. The *eosinophiles* show great absolute increase, and very often a relative increase,

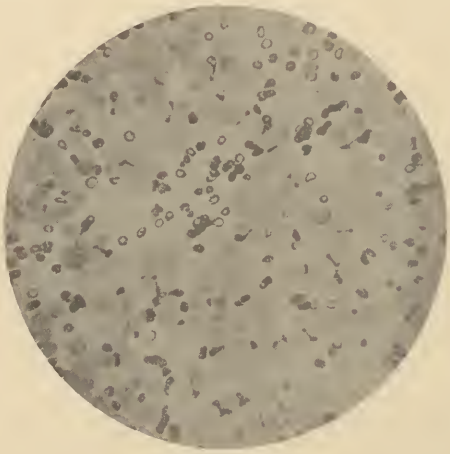

FIG. 119.—LEUKEMIA, WITH EXTREME INCREASE OF WHITE BLOOD-CELLS TO A COUNT OF ONE WHITE TO FOUR RED.

but it is the neutrophilic myelocyte which characterizes this form of the disease. They occur as normal, very small, and very large eosinophile cells.

### LYMPHATIC LEUKEMIA.

It is held by some observers that the structural differences in this form of the disease is owing to the rapidity of the alteration in the blood; but the changes are undoubtedly the same in prolonged cases, and a blood count even then shows extreme predominance of small lymphocytes which characterize the disease.

The *red cells* are somewhat lower in number than in the splenic-myelogenous type (about 2,700,000); the nucleated type of cell is

*rarely seen*, and when present in the extreme cases the megaloblasts may equal the normoblasts.

The *white cells* are increased to a much less degree than in the splenic-myelogenous type, a ratio of 1 : 40 being a fair average. *Lymphocytes* make up 90 per cent. of this increase, and they are mainly of the small variety, although every size may be formed, from 11 to 15 $\mu$ and the large may predominate. It will then be apparent that:

| Splenic-myelogenous. | Lymphatic. |
|---|---|
| Red cells, 3,100,000; nucleated cells, numerous. | Red cells, 2,700,000; nucleated form rare. |
| White cells, one to seven or eight. | One to 40. |
| Myelocytes form 40 per cent. of white corpuscles. | Lymphocytes form 90 per cent. Myelocytes and eosinophiles scanty. |

The blood examination is absolutely diagnostic. In Hodgkin's disease the symptoms are the same but the blood is normal. Tumors of the spleen may show leukocytosis, but myelocytes are not present. Of malignant disease, adenitis, hydronephrosis, chronic malaria, etc., the same may be said.

## HODGKIN'S DISEASE.

The diagnosis of this disease is impossible from clinical symptoms or postmortem findings without an examination of the blood and finding it *practically normal*. It will be seen that the blood evidence has its importance on the negative side. Many cases diagnosed as Hodgkin's disease are tubercular and syphilitic glandular hypertrophy, and it is difficult to make a diagnosis during life. There is a tendency at present to class this disease among the infectious diseases, while surgeons continue to consider it a form of sarcoma. The *blood* may show severe anemia in the later stages, and when inflammation occurs in the glands a decided leukocytosis may arise.

## CHANGES OF SPECIAL DISEASES.

**Pneumonia** may be diagnosed in its early stages, as opposed to typhoid, malaria, and grip, by the presence of a leukocytosis. The same is true of pneumonia in the very young and very old. The presence of leukocytes argues nothing as to prognosis, but their absence makes it very bad.

**Typhoid fever** leaves a postfebrile anemia. There is *no leukocytosis* during the disease, but complications cause a leukocytosis. There is an increased number of young leukocytes. The diagnostic value of the absence of leukocytosis in uncomplicated cases is great. Malaria and tuberculosis, with which typhoid is confused, have no leukocytosis, but the Plasmodium malariæ in the one and the bacilli or general symptoms of the other render a diagnosis possible.

In **diphtheria** a blood examination has no diagnostic value.

**Scarlet fever** has a decided leukocytosis. Eosinophiles are increased in favorable cases and absent in severe cases.

**Measles**, with which scarlet fever may be confused, has no leukocytosis.

**Septicemia** shows a rapid and severe anemia, a decided leukocytosis in even very mild cases, and the presence of pyogenic cocci.

In **appendicitis** leukocytosis will exclude colic, ovarian neuralgia, gall-stone and nephritic colic, and impaction of feces.

**Tuberculosis** has no leukocytosis.

**Syphilis** has a combination of leukocytosis in young cells, with an eosinophilia, which is important. A high percentage of young leukocytes, combined with low hemoglobin percentage, means a severe type of the disease, and marked anemia, with the presence of myelocytes in the tertian stage, renders the prognosis serious.

In **malignant disease** (carcinoma and sarcoma) there may be no blood changes with small, slow-growing tumors, but in advanced cases the red cells become pale, thin, and are diminished almost as much as in pernicious anemia; hemoglobin percentage is decreased; normoblasts and megaloblasts are present, but the former predominate. Leukocytosis is present.

# BLOOD PARASITES.

## Malaria.

The best time for finding the Plasmodium malariæ in the blood is eight hours before or after a chill, but for a study of their life history continuous examinations should be made. A specimen of blood is obtained by pricking the cleansed lobe of the ear with a needle, allowing two or three drops to exude, then a clean cover-glass is touched to the next drop and placed at once on a clean slide. A $\frac{1}{12}$-inch oil-immersion lens is used in the examination. All malarial *organisms* are found within the blood-cells alone.

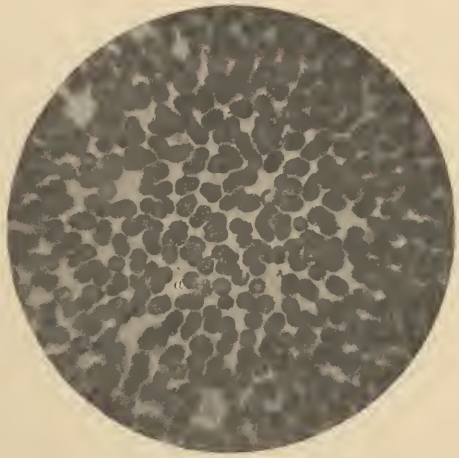

Fig. 120.—Pigmented Plasmodium Malariæ in the Stage of Segmentation. The single organism is seen below the center of the field (*a*).

The **Plasmodium malariæ** appear in the early stages of their growth as **hyaline forms**, which are light-colored, non-pigmented spots in the corpuscle. In order to distinguish these hyaline forms from clear spaces that appear abundantly in normal blood, it will be well to say that: (*a*) They are very few in number and are found with difficulty; (*b*) they are never in the center of a corpuscle; (*c*) they are irregular or branching in shape; (*d*) they grow dimmer or clearer in focusing, and not larger or smaller; (*e*) their edges fade off gradually; and (*f*) the hyaline body changes its form and position rapidly.

**Pigment granules**, with a very rapid and characteristic motion, soon appear in the hyaline body, and, as the paroxysm approaches,

this pigment works its way toward the center in radiating lines until it has collected in a black mass. The blood-corpuscle has by this time quite disappeared, and the body of the organism shows lines of division radiating from the center.

**Segmentation** now sets up along the lines of division, and a great number of spherical bodies result. It would seem that these small spherules would appear in the blood-plasma, but they are not seen again until they appear as hyaline bodies in other corpuscles.

Late in the life history of the organism **flagellæ**, or arms, suddenly appear on the surface of the plasmodium and lash about, producing a great disturbance in the surrounding corpuscles.

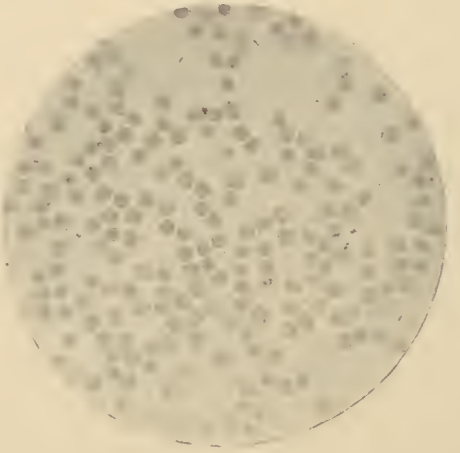

FIG. 121.—CRESCENTIC PLASMODIUM MALARIÆ.

These flagellæ are about 20 $\mu$ long and 2 $\mu$ wide, are perfectly transparent, or may have pigment distributed through them, especially in the tip. They sometimes fly off and dart free among the blood-corpuscles.

**Crescentic forms** of malarial organisms are always present in the estivo-autumnal forms of malaria after the first week. These are organisms with a crescentic body, usually lying on one side of, and decidedly larger in radius than, the red blood-cell, and have a mass of pigment collected in the middle of the crescent.

*Pigmented leukocytes* containing a part or an entire plasmodium are sometimes found in the blood near the time of the chill.

The blood shows either a simple anemia, an anemia identical

with pernicious anemia, where megaloblasts are present, or a progressive anemia, where continued destruction renders regeneration impossible. There is no leukocytosis.

### Filaria Sanguinis Hominis.

The Filaria sanguinis hominis is a blood parasite found mostly in the tropical regions, but occasionally in the United States. The blood of patients with chyluria or elephantiasis must be examined fresh after nine o'clock in the evening, inasmuch as the parasite wanders from the lymphatics into the blood only at night. It is the embryo of this parasite that we find in the blood. It is about $\frac{1}{80}$ of an inch in length, or 50 times the diameter of a red blood-cell, and about the width of such a cell. It has great vitality, and moves for a week under the cover-glass even though subjected to a low temperature. It is inclosed in a sheath, and tapers posteriorly for $\frac{1}{5}$ of its length to a very fine point. The head tapers but slightly, is rounded, and when alive a slight motion, as of breathing, is seen. In the middle of the body a granular collection is seen along its axis, but it is otherwise entirely transparent. It has no power of locomotion, but merely wriggles in one spot.

### Spirochæta (or Spirillum) of Relapsing Fever.

Obermeier has found in relapsing fever a constant organism which in length is about six times the diameter of a red blood-corpuscle, a mere thread in width, and twisted to form a corkscrew spiral. It has an active motion, especially in extension of the spiral, and can in this way increase its length greatly, but it also has a motion of undulation and actual locomotion. Their number is greatest at the beginning of a paroxysm ; their history is short ; they multiply very rapidly, and are absent between paroxysms.

They produce an anemia and a leukocytosis of the blood.

# APPENDIX.

### NOTE TO CAUSES OF TUMORS, PAGE 37.

Prof. H. Ribbert, in Zurich, considers Cohnheim's theory an insufficient explanation for the cause of tumors. He holds that every cell of every tissue has great intrinsic proliferative power of growth, and that, as a result of abnormalities of development, inflammation and connective-tissue growth between individual cells, certain cells of the tissue are loosened up from their contact with one another, thereby removing the physiological restraint which every cell exercises upon its neighbor. The static equilibrium of the tissues is set aside, and the cells "grow wild." Furthermore, he believes that the main element of danger in epithelial tissue is not necessarily downgrowth of cells, but that we are dealing with an upgrowth of misplaced epithelial cells.

### NOTE TO PAGE 37, OR TO PAGE 61.

Dr. H. W. Johnstone, of Cincinnati, concludes that all cells, epithelial and connective-tissue, spring directly from connective tissue; that the reticular tissue of the body, the sustentacular tissue of secreting glands, and the *stratum lucidum* of dermal and mucoid structures are closely allied in a definite function of furnishing the protoplasm which supplies the waste of the daily wear and tear; that these tissues are highly vitalized, definitely connected with and governed by, if not an actual part of, the sympathetic nervous system. That by trauma, inflammatory thickening, etc., the nervous control over these tissues may be withdrawn, and a limitless growth result. Furthermore, that this nervous control will explain the frequently observed antecedence of neurasthenia, and of overpowering nervous shock in cases of cancer.

### NOTE TO GLIOMA, PAGE 48.

Recent investigations in the nervous system seem to prove that no connective tissue exists in the central nervous system; that the neuroglia is of ectodermal (or epiblastic) origin, and that the glioma is an epithelial tumor.

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